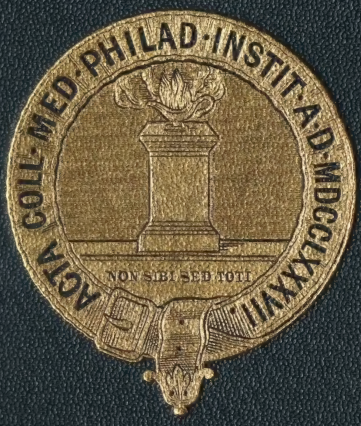
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OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA.

THIRD SERIES.

VOLUME THE FOURTEENTH.

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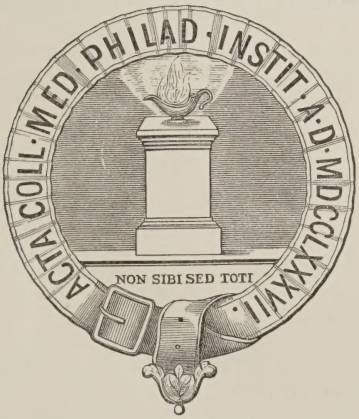

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The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its Transactions.

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1892.

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LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED

1787. JOHN REDMAN, M.D.

1805. WILLIAM SHIPPEN, M.D.

1809. ADAM KUHN, M.D.

1818. THOMAS PARKE, M.D.

1835. THOMAS C. JAMES, M.D.

1835. THOMAS T. HEWSON, M.D.

1848. GEORGE B. WOOD, M.D., LL.D.

1879. W. S. W. RUSCHENBERGER, M.D.

1883. ALFRED STILLÉ, M.D., LL.D.

1884. SAMUEL LEWIS, M.D.

1884. J. M. DA COSTA, M.D., LL.D.

1886. S. WEIR MITCHELL, M.D., LL.D.

1889. D. HAYES AGNEW, M.D., LL.D.

1892. S. WEIR MITCHELL, M.D., LL.D.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1892.

[Non-resident Fellows are marked *.]

ELECTED

1883. Abbot, Griffith E., Ph.D., M.D.

1892. Abbott, A. C., M.D., First Assistant Laboratory of Hygiene, University of Pennsylvania.

1870. Adler, John M., M.D.

1876. Alison, Robert H., M.D.

1867. Allen, Harrison, M.D., Professor of Zoölogy and Comparative Anatomy in the University of Pennsylvania.

1873. Allis, Oscar H., M.D., Clinical Lecturer on Orthopædic Surgery in Jefferson Medical College, and Surgeon to the Hospital of the same; Surgeon to the Presbyterian Hospital.

1888. Anders, James M., M.D., Professor of Hygiene and Clinical Diseases of Children in the Medico-Chirurgical College, Philadelphia; Physician to the Philadelphia Hospital.

1869. Andrews, T. Hollingsworth, M.D., Consulting Surgeon to the Hospital of the Good Shepherd, Radnor; Medical Director of the Bureaus of Police and Fire of the Department of Public Safety.

*1882. Ashbridge, Richard, M.D., Assistant Surgeon U. S. Navy.

1863. Ashhurst, John, Jr., M.D., Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and the Children's Hospitals; Consulting Surgeon to St. Christopher's and the Woman's Hospitals, and to the Hospital of the Good Shepherd, Radnor.

ELECTED

1865. Ashhurst, Samuel, M.D., Surgeon to the Children's Hospital.

1857. Atlee, Walter Franklin, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat.

1852. Bache, Thomas Hewson, M.D.

1883. Baer, Benjamin F., M.D., Professor of Gynecology in the Philadelphia Polyclinic.

1892. Baker, George Fales, B.S., M.D.

1879. Baker, Washington H., M.D., Obstetrician to the Maternity Hospital.

1876. Baldwin, Louis K., M.D., Examining Physician to the Hospital of the Good Shepherd, Radnor.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Gynecologist to St. Agnes' Hospital; Surgeon to Gynecean Hospital.

1880. Bartholow, Roberts, M.D., Professor (Emeritus) of Materia Medica, General Therapeutics, and Hygiene in Jefferson Medical College.

1883. Baum, Charles, M.D., A.M., Ph.D.

1873. Baxter, H. F., M.D.

1883. Beates, Henry, M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician to St. Christopher's Hospital for Children, and to Children's Seashore House, Atlantic City.

1884. Biddle, Alexander W., M.D.

1884. Biddle, Thomas, M.D.

*1866. Black, J. J., M.D.

*1867. Boardman, Charles H., M.D.

1859. Boker, Charles S., M.D., Surgeon to St. Joseph's Hospital.

1891. Boyd, George M., M.D., Physician to the Lying-in Charity; Surgeon to the Out-door Department Episcopal Hospital; Assistant Surgeon to the Kensington Hospital for Women.

1884. Bradford, Thomas Hewson, M.D., Physician to the Dispensary of the Children's Hospital and to the Gynecological Departments of the Pennsylvania and the Howard Hospitals.

ELECTED

1856. Brinton, John H., M.D., Professor of the Practice of Surgery and of Clinical Surgery in Jefferson Medical College; Surgeon to St. Joseph's Hospital; Consulting Surgeon to the Southwestern Hospital of Philadelphia.

1891. Brinton, Lewis, M.D.

1887. Brubaker, Albert P., M.D., Professor of Physiology and General Pathology in the Pennsylvania College of Dental Surgery; Demonstrator of Physiology in Jefferson Medical College; Lecturer on Anatomy and Physiology at the Drexel Institute.

*1890. Brush, Edward N., M.D., Medical Superintendent of the Sheppard Asylum, Sheppard, Md.

*1851. Bullock, William R., M.D.

1887. Bunting, Ross R., M.D.

1870. Burnett, Charles H., M.D., Clinical Professor of Otology in the Woman's Medical College; Professor (Emeritus) of Otology in the Philadelphia Polyclinic.

1892. Burr, Charles W., M.D., Visiting Physician to St. Joseph's Hospital, and the Home for Incurables; Visiting Pathologist to the State Asylum for the Insane.

1886. Cadwalader, Charles E., M.D.

1892. Cattell, Henry W., M.D.

*1892. Cerna, David, M.D., Ph.D., Demonstrator of Physiology in the Department of Medicine of the University of Texas; Corresponding Fellow of the Sociedad Española de Higiene of Madrid.

1885. Chapin, John B., M.D., Physician to the Pennsylvania Hospital for the Insane.

1880. Chapman, Henry C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in Jefferson Medical College.

1868. Cheston, D. Murray, M.D.

1873. Clark, Leonardo S., M.D.

1872. Cleemann, Richard A., M.D.

*1842. Clymer, Meredith, M.D.

1871. Cohen, J. Solis, M.D., Professor (Emeritus) of Diseases of the Throat and Chest in the Philadelphia Polyclinic; Professor (Honorary) of Laryngology in Jefferson Medical Col-

ELECTED

lege; Consulting Physician to the Home for Consumptives, Philadelphia.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine and Applied Therapeutics in the Philadelphia Polyclinic and Physician to the Polyclinic Hospital; Clinical Lecturer on Medicine in Jefferson Medical College; Visiting Physician to the Philadelphia Hospital; Consulting Physician to the Jewish Hospital.

1866. Cruice, R. B., M.D., Surgeon to St. Joseph's Hospital.

1884. Curtin, R. G., M.D., Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the University Hospital; Physician to the Philadelphia and Presbyterian Hospitals.

1884. Da Costa, John C., M.D., Gynecologist to Jefferson Medical College Hospital and to St. Agnes' Hospital.

1858. Da Costa, J. M., M.D., LL.D., Professor (Emeritus) of the Principles and Practice of Medicine in Jefferson Medical College; Physician to the Pennsylvania Hospital; Consulting Physician to the Children's Hospital and to the Northern Dispensary.

1887. Daland, Judson, M.D., Instructor in Clinical Medicine and Lecturer on Physical Diagnosis and Symptomatology in the University of Pennsylvania; Assistant Physician to the University Hospital; one of the Examiners of the Insane to the Philadelphia Hospital; Physician to the Rush Hospital for Consumption.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1888. Davis, Edward P., M.D., Professor of Obstetrics and Diseases of Children in the Philadelphia Polyclinic; Demonstrator of Obstetrics in Jefferson Medical College; Visiting Obstetrician to the Philadelphia Hospital.

1889. Davis, G. G., M.D., Assistant Surgeon to the Episcopal and Orthopædic Hospitals; Surgeon to the Dispensary of the Children's Hospital; Assistant Demonstrator of Surgery in the University of Pennsylvania.

1874. Deakyne, A. C., M.D.

*1870. Deal, L. J., M.D.

ELECTED

1887. Deaver, John B., M.D., Associate Professor of Anatomy in the University of Pennsylvania; Surgeon to the Philadelphia Hospital, to the German Hospital, and to St. Mary's Hospital.

1892. Deaver, Richard Wilmot, M.D.

1885. Dercum, Francis X., M.D., Clinical Professor of Neurology in Jefferson Medical College; Neurologist to the Philadelphia Hospital.

1891. Dixon, Samuel G., M.D., Professor of Microscopic Technology and Histology, and Curator of the Academy of Natural Sciences of Philadelphia.

1891. Dixon, William C., M.D., Physician to Industrial Home for Blind Women, Philadelphia; Physician to the Shelter for Colored Orphans, Philadelphia; Member of Consulting Staff, Philadelphia Home for Incurables; Examiner of Insane Patients, Philadelphia Hospital.

1884. Downs, R. N., M.D.

1884. Drysdale, T. M., M.D.

1864. Duer, Edward L., M.D., Accoucheur to the Philadelphia Hospital; Surgeon to the Maternity Hospital; Visiting Physician to the Preston Retreat.

1871. Duhring, L. A., M.D., Professor of Skin Diseases in the University of Pennsylvania.

1881. Dulles, Charles Winslow, M.D., Surgeon to Rush Hospital; Surgeon to Out-patients, Presbyterian Hospital; Medical Director of the Presbyterian Ministers' Fund.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D.

1888. Dunn, Thomas D., M.D.

*1849. Dunnott, Justus, M.D.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

1882. Edwards, Joseph F., M.D.

*1887. Edwards, William A., M.D.

*1880. Eskridge, J. T., M.D.

1868. Evans, Horace Y., M.D., Physician to the Charity Hospital.

1884. Fenton, Thomas H., M.D.

ELECTED

1866. Fischer, Emil, M.D.

1884. Fisher, Henry M., M.D., Physician to the Episcopal Hospital; Microscopist to the Pennsylvania Hospital, and Physician to the Out-patient Department of the same.

1888. Flick, Lawrence F., M.D.

1862. Forbes, William S., M.D., Professor of Anatomy in Jefferson Medical College.

1870. Ford, William H., M.D., President of the Board of Health; Physician to the Foster Home.

*1885. Fox, Joseph M., M.D.

1890. Freeman, Walter J., M.D., Clinical Assistant to the Throat Department of the Philadelphia Polyclinic.

1885. Fricke, Albert, M.D.

1889. Fussell, M. Howard, M.D., Chief Physician to the Medical Dispensary of the University of Pennsylvania; Instructor of Clinical Medicine in the University of Pennsylvania.

1873. Gerhard, George S., M.D.

1884. Getchell, F. H., M.D.

1892. Gibb, Joseph S., M.D.

1885. Girvin, Robert M., M.D., Gynecologist to the Presbyterian Hospital.

1889. Githens, William H. H., M.D., Visiting Physician to "The Sheltering Arms."

1884. Godey, Harry, M.D.

1868. Goodell, William, M.D., Professor of Clinical Gynecology in the University of Pennsylvania; Consulting Physician to the Lying-in Department of the Northern Dispensary.

1867. Goodman, H. Ernest, M.D., Professor of Surgery in the Medico-Chirurgical College; Surgeon to Wills Eye Hospital and to the Orthopædic Hospital; Consulting Surgeon to the Maternity Hospital.

1885. Graham, John, M.D.

1891. Green, Walter D., A.M., M.D., Out-patient Surgeon to the Pennsylvania, Children's, and Methodist Hospitals; Assistant Surgeon to the Gynecean Hospital.

1870. Grier, M. J., M.D.

1883. Griffith, J. P. Crozer, M.D., Clinical Professor of the Diseases of Children in the Hospital of the University of Penn-

ELECTED

sylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to St. Agnes', the Children's, and the Howard Hospitals.

1871. Grove, John H., M.D., Surgeon to St. Mary's and to St. Agnes' Hospitals.

1889. Guiteras, John, M.D., Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania.

1863. Hall, A. Douglass, M.D., Surgeon to Wills Eye Hospital.

1890. Hall, John C., M.D.

*1859. Hammond, William A., M.D., Surgeon-General U. S. A. Retired.

1886. Hansell, Howard F., M.D., Chief Clinical Assistant to the Ophthalmological Department of Jefferson Medical College Hospital; Ophthalmic and Aural Surgeon to the Southwestern Hospital.

1889. Hare, Hobart A., M.D., Professor of Therapeutics in Jefferson Medical College; Physician to St. Agnes' Hospital and to Jefferson Medical College Hospital.

1865. Harlan, George C., M.D., Surgeon to Wills Eye Hospital and to the Eye and Ear Department of the Pennsylvania Hospital; Professor (Emeritus) of Diseases of the Eye in the Philadelphia Polyclinic.

1863. Harlow, Lewis D., M.D.

1862. Harris, Robert P., M.D.

1885. Harte, Richard H., M.D., Demonstrator of Osteology in the University of Pennsylvania, and Assistant Surgeon to the Hospital; Surgeon to the Out-patient Department of the Pennsylvania Hospital.

1851. Hartshorne, Henry, M.D., LL.D.

1888. Hartzell, Milton B., M.D., Pathologist to the Presbyterian Hospital; Assistant Physician to the Dispensary for Skin Diseases, University of Pennsylvania.

*1849. Hastings, John, M.D.

1872. Hays, I. Minis, M.D.

1882. Hearn, W. Joseph, M.D., Surgeon to the Hospital of Jefferson Medical College and to the Philadelphia Hospital.

1884. Henry, Frederick P., M.D., Physician to Jefferson Medical College Hospital and to the Philadelphia Hospital; Pro-

ELECTED

fessor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

1891. Hewson, Addinell, A.M., M.D., Demonstrator of Anatomy in Jefferson Medical College; Chief Clinical Assistant in the Surgical Dispensary of Jefferson Medical College Hospital; Dispensary Surgeon to the Hospital of the Protestant Episcopal Church.

1872. Hinkle, A. G. B., M.D.

1892. Hinsdale, Guy, M.D., Lecturer on Climatology in the University of Pennsylvania; Physician to the Presbyterian Orphanage and to the Out-patient Department of the Presbyterian Hospital; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases.

1888. Hirsh, Abram B., M.D.

1888. Hirst, Barton Cooke, M.D., Professor of Obstetrics in the University of Pennsylvania; Obstetrician to the Philadelphia Hospital and to the Maternity Hospital.

1885. Holland, James W., M.D., Professor of Medical Chemistry and Toxicology in Jefferson Medical College.

1879. Hopkins, William Barton, M.D., Surgeon to the Episcopal Hospital and to the Out-patient Department of the Pennsylvania Hospital.

1867. Horn, George H., M.D., Professor of Entomology in the Biological Department of the University of Pennsylvania.

1888. Horwitz, Orville, M.D., Demonstrator of Surgery in Jefferson Medical College; Chief of the Out-door Surgical Department of Jefferson Medical College Hospital; Surgeon to the Philadelphia Hospital.

1868. Howell, Samuel B., M.D., Professor of Chemistry in the Medico-Chirurgical College.

1892. Hughes, Wm. E., M.D.

*1881. Huidekoper, Rush Shippen, M.D.

1884. Hunt, J. Gibbons, M.D.

1854. Hunt, William, M.D., Surgeon to the Pennsylvania Hospital.

1871. Ingham, James V., M.D.

1885. Jackson, Edward, M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Surgeon to Wills Eye Hospital; Ophthalmologist to Rush Hospital.

ELECTED

1887. Jayne, Horace, M.D., Professor of Vertebrate Morphology in the Biological Department of the University of Pennsylvania.

1885. Judd, Leonardo Da Vinci, M.D.

1867. Judson, Oliver A., M.D.

1886. Jurist, Louis, M.D., Chief Clinical Assistant in the Laryngological Department of Jefferson Medical College Hospital.

*1877. Keating, John M., M.D.

1849. Keating, William V., M.D., Physician to St. Joseph's Hospital.

1867. Keen, William W., M.D., LL.D., Professor of the Principles of Surgery and of Clinical Surgery in Jefferson Medical College; Surgeon to Jefferson Medical College Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes Hospital.

*1887. Kelly, Howard A., M.D., Professor of Gynecology in the Johns Hopkins University, and Gynecologist and Obstetrician to the Hospital.

*1844. King, Charles R., M.D.

1875. Kirkbride, Joseph J., M.D.

1892. Lainé, Damaso T., M.D.

*1865. La Roche, C. Percy, M.D.

1887. Leaman, Henry, M.D.

1883. Leffmann, Henry, M.D., Professor of Chemistry in the Philadelphia Polyclinic and in the Woman's Medical College; Pathological Chemist to Jefferson Medical College Hospital.

1892. Leidy, Joseph, M.D.

1855. Lewis, Francis W., M.D.

1877. Lewis, Morris J., M.D., Physician to the Children's Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to the Pennsylvania Hospital.

1886. Lloyd, J. Hendrie, M.D., Physician to the Nervous and Insane Department of the Philadelphia Hospital, to the Methodist Episcopal Hospital, and to the Home for Crippled Children.

ELECTED

1877. Longstreth, Morris, M.D., Professor of Pathological Anatomy in Jefferson Medical College; Physician to the Pennsylvania Hospital.

1886. MacCoy, Alexander W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Lecturer on Diseases of the Throat and Nose in the Woman's Medical College of Pennsylvania.

1875. McClellan, George, M.D., Surgeon to the Howard Hospital.

1871. McFerran, J. A., M.D.

*1885. Mallet, John W., M.D.

1889. Martin, Edward, M.D., Surgeon to the Howard Hospital; Clinical Professor of Genito-Urinary Surgery to the Hospital of the University of Pennsylvania.

1887. Massey, Isaac, M.D., Surgeon to the Pennsylvania Railroad.

*1850. Mayer, Edward R., M.D.

1885. Mays, Thomas J., M.D., Professor of Diseases of the Chest and of Experimental Therapeutics in the Philadelphia Polyclinic; Visiting Physician to Rush Hospital.

1868. Mears, J. Ewing, M.D., Professor of Anatomy and Clinical Surgery in the Pennsylvania College of Dental Surgery; Gynecologist to Jefferson Medical College Hospital; Surgeon to St. Agnes' Hospital.

1875. Meigs, Arthur V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind.

*1884. Mifflin, Houston, M.D.

1881. Mills, Charles K., M.D., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Clinical Professor of Mental Diseases in the University of Pennsylvania, and of Nervous Diseases in the Woman's Medical College; Neurologist to the Philadelphia Hospital, and Consulting Physician to the Department for the Insane of the Philadelphia Hospital.

1888. Mitchell, John K., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Physician to St. Agnes'

ELECTED

Hospital; Assistant Physician to the University Hospital and to the Infirmary for Nervous Diseases.

1856. Mitchell, S. Weir, M.D., Professor of Diseases of the Mind and Nervous System in the Philadelpha Polyclinic; Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Physician to the Maternity Hospital.

1882. Montgomery, Edward E., M.D., Clinical Professor of Gynecology in the Jefferson Medical College; Obstetrician to the Philadelphia Hospital.

1863. Morehouse, George R., M.D., Ph.D., Physician to St. Joseph's Hospital.

1886. Morris, Caspar, M.D., Physician to the Episcopal Hospital and to the Out-patient Department of the Pennsylvania Hospital.

1883. Morris, Henry, M.D., Gynecologist to the Howard Hospital.

1856. Morris, J. Cheston, M.D.

1861. Morton, Thomas G., M.D., Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to the Jewish Hospital; Emeritus Surgeon to Wills Eye Hospital.

1891. Morton, T. S. K., M.D., Professor of Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to the Polyclinic Hospital; Assistant Surgeon to the Orthopædic Hospital.

1864. Moss, William, M.D.

1890. Müller, Auguste F., M.D., Attending Physician to the Germantown Hospital.

1882. Musser, John H., M.D., Assistant Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Philadelphia Hospital and to the Presbyterian Hospital; Consulting Physician to the Woman's Hospital of Philadelphia and to the West Philadelphia Hospital for Women.

1886. Neff, Joseph F., M.D.

1887. Neilson, Thomas Rundle, M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-Urinary Diseases in the Philadelphia Polyclinic; Lecturer on Diseases of the Rectum, and Assistant Demonstrator of Anatomy in the University of Pennsylvania.

ELECTED

1889. Noble, Charles P., M.D., Surgeon-in-Chief to the Kensington Hospital for Women; Surgeon-in-charge of the Department for Women of the Northern Dispensary; Surgeon-in-charge of the Department for Women of the Union Dispensary; Lecturer on Gynecology in the Philadelphia Polyclinic.

1869. Norris, Herbert, M.D., Supervising Physician to St. Clement's Hospital.

1865. Norris, Isaac, Jr., M.D.

1892. Norris, Richard C., M.D., Demonstrator of Obstetrics, University of Pennsylvania; Assistant Obstetrician, University Maternity; Obstetric Registrar, Philadelphia Hospital; Visiting Physician to Methodist Hospital; Consulting Obstetrician and Attending Gynecologist to Southeastern Dispensary.

1866. Norris, William F., M.D., Honorary Professor of Ophthalmology and Clinical Professor of Diseases of the Eye in the University of Pennsylvania; Surgeon to Wills Eye Hospital.

1884. Oliver, Charles A., M.D., Attending Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Presbyterian Hospital; Consulting Ophthalmic Surgeon to St. Agnes', St. Timothy's, and the Maternity Hospitals.

1884. O'Neill, J. W., M.D.

*1885. Osler, William, M.D., Professor of Medicine in Johns Hopkins University, and Physician to the Hospital.

1890. Packard, Frederick A., M.D., Visiting Physician to the Episcopal and Methodist Hospitals; Physician to the Outpatient Department of the Pennsylvania and Children's Hospitals; Instructor in Physical Diagnosis in the University of Pennsylvania.

1858. Packard, John H., M.D., Surgeon to the Pennsylvania Hospital and to St. Joseph's Hospital.

1864. Pancoast, William H., M.D., Professor of Anatomy and of Clinical Surgery in the Medico-Chirurgical College; Consulting Surgeon to the Philadelphia Hospital for Skin Diseases.

ELECTED

1882. Parish, William H., M.D., Professor of Obstetrics in the Dartmouth Medical College; Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Obstetrician to the Lying-in Charity; Consulting Surgeon to the Kensington Hospital; Consulting Gynecologist to St. Agnes Hospital.

1883. Parvin, Theophilus, M.D., Professor of Obstetrics and Diseases of Women and Children in Jefferson Medical College.

1889. Penrose, Charles Bingham, M.D., Surgeon to the Gynecean Hospital; Surgeon to the Out-patient Department of the Pennsylvania and St. Agnes' Hospitals.

1854. Penrose, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania; Consulting Obstetrician to the Maternity Hospital; Visiting Physician to the Preston Retreat.

1868. Pepper, William, M.D., LL.D., Provost of the University of Pennsylvania, and Professor of the Theory and Practice of Medicine in the same.

1884. Perkins, Francis M., M.D., Ophthalmic and Aural Surgeon to the Dispensary of St. Mary's Hospital; Visiting Ophthalmic Surgeon to the Hospital of the Good Shepherd at Radnor.

1890. Phillips, J. Willoughby, M.D.

1883. Piersol, George A., M.D., Professor of Anatomy in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital and to the Philadelphia Hospital.

1885. Potter, Thomas C., M.D.

1887. Price, Jacob, M.D.

1889. Price, Joseph, M.D., Physician-in-charge of the Preston Retreat and of the Female Department of the Philadelphia Dispensary.

1889. Randall, B. Alexander, M.D., Professor of Otology in the University of Pennsylvania and in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's Hospital.

ELECTED

1887. Reed, Charles H., M.D.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania.

1888. Rex, George A., M.D.

1883. Rex, Oliver P., M.D., Clinical Lecturer on Diseases of Children in Jefferson Medical College, and Physician to the Hospital; Physician to the Presbyterian Hospital.

1891. Rhoads, Edward G., M.D.

1891. Risley, S. D., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Attending Surgeon at Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine.

1882. Roberts, A. Sydney, M.D.

1878. Roberts, John B., M.D., Professor of Anatomy and Surgery in the Philadelphia Polyclinic; Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Methodist Hospital.

1888. Robins, Robert P., M.D., Visiting Physician to the Dispensary of the House of Industry, to the Church Home for Children, and to the Board of Guardians of the Poor; Lecturer on Chemistry in the Episcopal Academy.

1838. Ruschenberger, W. S. W., M.D., Medical Director, U. S. N.

*1852. Sargent, Fitz William, M.D.

*1864. Sargent, Winthrop, M.D.

1866. Schaffer, Charles, M.D., Professor of Botany in the Pennsylvania Horticultural Society.

1887. De Schweinitz, George E., M.D., Clinical Professor of Ophthalmology in Jefferson Medical College; Professor of Ophthalmology in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's Hospital; Ophthalmologist to the Orthopædic Hospital and to the Philadelphia Hospital; Consulting Ophthalmic Surgeon to the Methodist Episcopal Hospital.

1892. Seiss, Ralph W., M.D., Adjunct Professor of Otology in the Philadelphia Polyclinic.

1888. Seltzer, Charles M., M.D.

1875. Seyfert, Theodore H., M.D.

ELECTED

1884. Shaffner, Charles, M.D., Ophthalmic Surgeon to the Presbyterian Hospital; Surgeon to Pennsylvania Eye and Ear Infirmary.

1887. Shakespeare, Edward O., M.D., Pathologist to the Philadelphia Hospital.

1876. Shippen, Edward, A.M., M.D., U. S. N. (retired).

1891. Shober, John B., M.D., Surgeon to the University Hospital Dispensary and to the Gynecean Hospital Dispensary; Examining Surgeon for Pensions, Philadelphia.

1890. Shoemaker, George Erety, A.M., M.D., Visiting Surgeon to the Methodist Hospital; Surgeon to Out-patient Department of the Presbyterian Hospital.

1880. Simes, J. H. C., M.D., Professor of Genito-Urinary and Venereal Diseases in the Philadelphia Polyclinic; Surgeon to the Episcopal Hospital and to St. Christopher's Hospital.

1873. Simpson, James, M.D., Physician to St. Mary's Hospital.

1872. Sinkler, Wharton, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the Philadelphia Hospital; Physician to the Epileptic Hospital of Philadelphia.

*1863. Smith, A. K., M.D., U. S. A.

*1864. Smith, Edward A., M.D.

1875. Starr, Louis, M.D., Physician to the Children's Hospital; Consulting Physician to the Maternity Hospital.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia Hospital and to the Jewish Hospital of Philadelphia.

1884. Stelwagon, Henry W., M.D., Ph.D., Clinical Professor of Dermatology in the Woman's Medical College; Clinical Professor of Dermatology in Jefferson Medical College; Dermatologist to the Philadelphia Hospital; Physician to the Department for Skin Diseases of the Howard Hospital and the Northern Dispensary.

1888. Stewart, David D., M.D., Clinical Lecturer on Medicine in the Jefferson Medical College; Physician to St. Mary's Hospital and to St. Christopher's Hospital for Children.

1842. Stillé, Alfred, M.D., LL.D., Professor (Emeritus) of the

ELECTED

Theory and Practice of Medicine in the University of Pennsylvania; Consulting Physician to the Maternity Hospital and to the Woman's Hospital.

1846. Stocker, Anthony E., M.D.

1884. Stryker, Samuel S., M.D.

1886. Taylor, John Madison, M.D., Physician to Howard Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Dispensary of the Children's Hospital; Professor of Children's Diseases in the Philadelphia Polyclinic.

1867. Taylor, R. R., M.D.

1887. Taylor, William J., M.D., Surgeon to St. Agnes' Hospital, and Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1886. Taylor, William L., M.D., Instructor in Clinical Gynecology in the University of Pennsylvania, and Chief of the Clinic and Assistant Gynecologist to the Hospital of the same; Surgeon-in-Chief to the Beacon Service for Women.

1867. Thomas, Charles H., M.D.

1869. Thomson, William, M.D., Professor (Honorary) of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Hospital of the same; Emeritus Surgeon to the Wills Eye Hospital.

*1854. Tilden, W. P., M.D.

*1870. Turner, A. Paul, M.D.

1866. Tyson, James, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University Hospital and to Rush Hospital for Consumption.

1864. Vandyke, E. B., M.D.

1873. Van Harlingen, Arthur, M.D., Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Howard Hospital.

1883. Vinton, Charles Harrod, M.D.

1885. Walker, James B., M.D., Attending Physician to the Philadelphia Hospital; Lecturer on Clinical Medicine and Consulting Physician to the Woman's Hospital.

ELECTED

1886. Watson, E. W., M.D.

1875. Webb, William H., M.D.

1883. Welch, William M., M.D., Physician to the Municipal Hospital for Contagious Diseases; Lecturer on Exanthemata and Vaccinia in the Medico-Chirurgical College.

1884. Wharton, Henry R., M.D., Demonstrator of Surgery in the University of Pennsylvania, and Assistant Surgeon to the Hospital of the University of Pennsylvania; Surgeon to the Children's, Presbyterian, and Methodist Hospitals.

1883. Whelen, Alfred, M.D.

1878. White, J. William, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Maternity Hospital.

1880. Willard, DeForest, M.D., Clinical Professor of Orthopædic Surgery in the University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to the White and to the Colored Cripples' Homes and to the Home for Incurables.

*1878. Williamson, Jesse, M.D.

1881. Wilson, H. Augustus, M.D., Professor of General and Orthopædic Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Clinical Professor of Orthopædic Surgery in the Woman's Medical College of Pennsylvania; Clinical Professor of Orthopædic Surgery in Jefferson Medical College; Consulting Surgeon to the Kensington Hospital for Women.

1874. Wilson, James C., M.D., Professor of the Practice of Medicine and of Clinical Medicine in Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician to the German Hospital.

1884. Wirgman, Charles, M.D., Physician to the Hospital of Jefferson Medical College and to the Howard Hospital.

1852. Wister, Owen Jones, M.D., Consulting Surgeon to the Germantown Hospital.

1865. Wood, Horatio C., M.D., Professor of Materia Medica, Pharmacy, and General Therapeutics in the University of Pennsylvania, and Clinical Professor of Diseases of the Nervous System in the Hospital of the same.

ELECTED

1880. Woodbury, Frank, M.D., Honorary Professor of Clinical Medicine in the Medico-Chirurgical College of Philadelphia, and Physician to the Hospital of the same.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1888. Woodward, Charles E., M.D., Physician to the Chester County Prison and West Chester Board of Health; U. S. Examining Surgeon.

1878. Wormley, Theodore G., M.D., LL.D., Professor of Chemistry in the University of Pennsylvania; U. S. Examining Surgeon.

1860. Wurts, Charles Stewart, M.D.

1861. Yarrow, Thomas J., M.D.

1889. Young, James K., M.D., Instructor in Orthopædic Surgery and Assistant Demonstrator of Surgery in the University of Pennsylvania; Orthopædic Surgeon in the Out-patient Department of the Hospital.

1887. Ziegler, Walter M. L., M.D., Assistant Aural Surgeon and Chief of the Dispensary for Diseases of the Ear in the Hospital of the University of Pennsylvania.

[It is particularly requested that any change of appointment, or any error in the titles of Fellows as published, may be communicated to the Committee of Publication before the first of November in each year, in order that the list may be made as nearly correct as possible.]

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

ELECTED

1873. Ackland, Henry W., M.D., F.R.S., Oxford, England.
1890. Bacelli, Guido, Rome, Italy.
1877. Barnes, Robert, M.D., London, England.
1876. Billings, John S., M.D., U. S. A., Washington, D. C.
1886. Bowditch, Henry P., M.D., Boston, Massachusetts.
1865. Butcher, R. G., M.D., M.R.C.S., Dublin, Ireland.
1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.
1886. Cheever, David W., M.D., Boston, Massachusetts.
1876. Comegys, C. G., M.D., Cincinnati, Ohio.
1876. Corson, Hiram, M.D., Norristown, Pennsylvania.
1876. Davis, N. S., M.D., Chicago, Illinois.
1886. Draper, William H., M.D., New York.
1892. Emmet, Thomas Addis, M.D., New York.
1883. Fayrer, Sir Joseph, M.D., LL.D., F.R.S., London, England.
1892. Fitz, Reginald H., M.D., Boston, Mass.
1876. Green, Traill, M.D., Easton, Pennsylvania.
1883. Heath, Christopher, F.R.C.S., London, England.
1892. Holmes, Oliver Wendell, M.D., Boston, Mass.
1874. Jackson, J. Hughlings, M.D., London, England.
1891. Jacobi, A., M.D., New York.
1876. Johnson, George, M.D., F.R.S., London, England.
1876. Jones, Joseph, M.D., New Orleans, Louisiana.
1876. King, James, M.D, Pittsburg, Pennsylvania.
1877. Lister, Sir Joseph, Bart, M.D., LL.D., F.R.S., London, England.
1865. Macleod, G. H. B., M.D., Glasgow, Scotland.

ELECTED

1886. McGuire, Hunter, M.D., Richmond, Virginia.
1876. Moore, E. M., M.D., Rochester, New York.
1876. Mowry, R. B., M.D., Allegheny City, Pennsylvania.
1873. Ogle, John W., M.D., London, England.
1874. Paget, Sir James, Bart, M.D., LL.D., F.R.S., D.C.L., London, England.
1876. Pollock, A. M., M.D., Pittsburg, Pennsylvania.
1876. Porcher, F. Peyre, M.D., Charleston, South Carolina.
1886. Reeve, John C., M.D., Dayton, Ohio.
1886. Senn, Nicholas, M.D., Milwaukee, Wisconsin.
1886. Shattuck, George C., M.D., Boston, Massachusetts.
1886. Thomas, T. Gaillard, M.D., New York.
1869. Valcourt, Th. de, M.D., Cannes, France.
1892. Virchow, Rudolf, M.D., Berlin, Germany.
1892. Welch, William H., M.D., Baltimore, Maryland.
1886. Whittaker, James T., M.D., Cincinnati, Ohio.
1886. Yandell, David W., M.D., Louisville, Kentucky.

CORRESPONDING MEMBERS.

ELECTED

1880. Carrow, Fleming, M.D., United States.
1880. Chiara, Domenico, M.D., Florence, Italy.
1886. Dey, Kanny Loll, M.D., Calcutta, India.
1885. Rendu, Jean, M.D., Lyons, France.
1886. Richards, Vincent, Goalunda, India.
1889. Strahan, John, M.D., Belfast, Ireland.

NECROLOGICAL LIST FOR 1892.

Fellows.

D. HAYES AGNEW.
HENRY F. FORMAD.
JOHN J. REESE,
TOBIAS G. RICHARDSON (non-resident).
ELISHA B. SHAPLEIGH.

Associate Fellow.

HENRY I. BOWDITCH.

Corresponding Member.

GREGORIO FEDELI.

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FEB 24 1920

THE WILLIAM F. JENKS MEMORIAL PRIZE.

The William F. Jenks Memorial Prize for 1889 was awarded to Dr. John Strahan, of Belfast, Ireland, for his Essay on the "Diagnosis and Treatment of Extra-Uterine Pregnancy."

ALVARENGA PRIZE.

The Alvarenga Prize for 1892 has been awarded to Dr. R. H. L. Bibb, of Saltillo, Mexico, for his Essay entitled "Some Observations on the Nature of Leprosy."

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THE PRESIDENT'S ADDRESS.

By S. WEIR MITCHELL, M.D., LL.D.

[Read December, 1892.]

The continuous prosperity of an institution makes the annual task of the presidential address by no means easy. Your various committees fully cover in their reports the year's business, and I have only to congratulate you upon the constant fidelity with which their various duties are performed. As time goes on, the College has had, as to its different modes of active usefulness, most notable changes. When first I knew it, our books were in two or three small cases; to-day, this collection is in the front rank of great medical libraries. Of its priceless value to every medical scholar in the city I need not speak. It is beginning to know and feel the risks and wants which come with enlarging growth. Nowadays, whole sciences arise newborn within the great area of medicine, and have, like bacteriology, a literature and journals of their own. All such novel claims have to be considered. Amidst the vast increase in the product of books, theses, and magazines, we are obliged to choose with discreet care what we shall buy and what reject. In the future we may, alas! have reason to repent many of these rejections. Also, it is necessary to spend a moderate amount in the purchase of old, and even of rare books, for the chance to obtain these lessens as years go by. "A great library," said Mr. Talcott Williams, when opening the library of the University, "is the memory of mankind;" and a great medical library should, so to speak, remember on its shelves not merely to-day and yesterday, but the centuries gone by—their folly and their wisdom. This is not to be only a circulating library, with what I may call the light literature of the day; it must, of course, fulfil the daily needs of the prac-

titioner, and give him the journals and the fast-coming monographs; but the scholar also should here find the unusual books, the dusty tomes in which the history of the mind of medicine is so voluminously written. We are, I grieve to say, very far from satisfying this higher need. I asked Dr. Billings how nearly complete was the Surgeon-General's collection. I gathered from what he said in reply that its 85,000 volumes make it the most complete representation in library form of any single form of human learning. It needs about 8000 or 9000 attainable books. There will then be left about 1200 books—incunabula, rare editions, curios—which cannot be purchased. Ten thousand dollars are needed every year to keep it up to the times, and this exclusive of salaries. We have here some 45,000 books, and expend in all about six thousand dollars a year.

Yet it is characteristic of the chance peculiarities of libraries that last year I found here books not in the larger collection, and that despite its size there were twenty-five books, chiefly Italian, which were neither there nor anywhere else in America. You see, therefore, how large, how varied, how expensive, are the wants of a great library. When, a few years ago, the generosity and foresight of a woman induced the Fellows doubtfully to consent to the Nurse Directory, it could hardly have been suspected that not only was this bureau to be of immense value to the community, but that the very existence, the active existence of this library, would depend upon the increasing aid given by the Directory in its annual contributions, which in 1892 reached the sum of $2700.

To this increase and its happy resultant help there is, of course, a limit. Yet you must in reason expect your library to make even larger demands for money. It is an expensive necessity.

An effort should be made to obtain an endowment by appealing to the laity. It sometimes happens that fortunate opportunities in this direction present themselves to the Fellows, and I beg of you, therefore, to lose no chance of thus profitably serving the College.

Sections. The question of sections will again come before

you this evening, and before final settlement it has seemed desirable to learn the opinions of a larger number of Fellows than can attend our meetings—a plan of effecting this will be laid before you.

In the last year we have lost from our roll by death nine members. Of these, four were Associate Fellows, one a Corresponding Member, and one a non-resident Fellow. There has been one resignation. Thirteen Fellows have been elected, and five Associate Fellows, so that the active College now numbers 241 resident members.

Among those taken from us by death was D. Hayes Agnew, lately our President. The admirable portrait which faces me very fairly expresses the gravity, the dignity, and the tranquil good sense of this great surgeon. He fitly continued the long succession of notable men who have held the honors of this chair, and he was not unmindful in death of this institution, to which he had a warm and loyal attachment. He was the first surgeon to become President since Th. T. Hewson, 1835 to 1848.

Prof. J. J. Reese died September 4, 1892. He had a long and somewhat varied career as a teacher, and many years ago gave up general practice to devote himself to toxicology and legal medicine, which he taught with assiduity and success at the University of Pennsylvania for many years.

Prof. Formad, who died June 5th, was one of those who, on account of contributions of value to medical science, received the honor of Fellowship without payment of dues. Prof. Leidy had also this privilege, and the Fellows should remember that it is thus possible to reward and recognize such services when it becomes desirable so to do.

Tobias G. Richardson, a non-resident Fellow, long a practitioner in New Orleans, must easily live in remembrance for many of us, but I have failed to receive either as to him, as to Dr. Frank Donaldson, or to Dr. Kinloch, such information as would enable me to speak of them as I should have desired to do.

It has not been our custom to read memoirs of our Associate Fellows who die, hence it is well that I remind you of the

unusual eminence of those we have lost this year. Of Henry I. Bowditch no one who knew him can speak without delightful recollections of a noble presence, of gracious and courteous manners and intellectual powers, to which all social graces lent their ready charm. He seemed to me to represent a class found nowhere else than in our own great American cities, because nowhere else do men of the highest social position look upon a career in medicine as equal in honor to any which is open to the ambition of men. Of Dr. Bowditch's contributions to medicine there is no need to speak. They are familiar to all of us, and are among the substantial gains of the medicine of this century.

Prof. Gregorio Fedeli, of Rome, who died October, 1892, was one of our corresponding members; he justified his title by several valuable contributions to our volumes within the last few years. Prof. Fedeli was in the higher ranks of Italian professional life, and was medically active in so many ways that he was rewarded with numerous evidences of the appreciation of his brethren and of his government. His gracious hospitality to me and to others of the profession were made the more agreeable by the charm of kindly manners and large general cultivation.

Prof. Christopher Johnston, of Baltimore, died in 1891, within our official year. This distinguished surgeon was, of all in our year's list of deaths, personally the best known to me—almost a life-long friendship has left with me the memory of a most upright and high-spirited man. Accurate and thoughtful, he used with conscientious care the great surgical powers which owed a part of their success to the extraordinary manual dexterity of which he was the master. A gentle and affectionate disposition won and kept for him the regard of the profession and of the many friends who, like myself, regret his loss as something distinct and valuable taken out of life.

You will, I know, share with me the regret I own at the resignation of the treasurership by Dr. Charles S. Wurtz. Of the punctuality, the care, the intelligence, and the never-failing courtesy with which he has filled this office, I cannot too warmly speak.

A RARE TYPE OF MALARIAL FEVER.

By COMMENDATORE GREGORIO FEDELI, M.D.,

(CORRESPONDING FELLOW)

LATE PHYSICIAN TO THE HOSPITAL OF STO. SPIRITO, AND IN EXTRAORDINARY OF S. GIO. DI DIO; PRESIDENT OF THE MEDICAL COMMITTEE OF EXAMINATION; SENIOR PHYSICIAN OF THE ORDER OF MALTA; LATE PHYSICIAN OF THE CITY OF ROME; TREASURER OF THE ROYAL ACADEMY OF MEDICINE OF ROME, ETC.

[Read January 6, 1892.]

GLANCING over the medical literature of the past, I find that Borsieri in his treatise upon *Febribus Intermittentibus*, and also Joseph Franck in his bibliography, call to mind how writers of various epochs, from the time of Hippocrates, have not failed to observe and describe the types of intermittent malarial fevers that recur at long intervals. In fact Borsieri, in his celebrated work (Cap. 65), notes that besides intermittent fevers of the quotidian, tertian, and quartan character which are more commonly met with in practice, quintidian, sextidian, septidian and nonidian types were observed by Hippocrates and others. Fevers recurring at longer intervals were also described by not a few other authors of antiquity, whose names I omit for the sake of brevity. That which is important for my purpose is to remember how Gentilis, upon the testimony of Nicolo Fiorentino, Rases, Ballonio, Nignisolio, and Werlofio, asserts that fevers of the type *quatuordecimanam* and *quindecimanam* have been observed "Nec de his dubium ullum unquam superesse potest, utrum certa et restanti periodo vere talis sese ostenderit."

In recent treatises on medical pathology, so far as I know, there is not a word of recent observations of such types of intermittent malarial fevers. Valleix, writing about them,

mentions, as a matter of history only, the existence of slow fevers; thinking perhaps that they are caused by a new infection, and do not belong to the genuine type. Likewise in the encyclopædic articles they are historically recorded, as noticed by our fathers, and nothing more. Neither from Italian nor from foreign journals could I collect recent facts bearing upon my argument; nor yet in my private practice have I, until recently, had the opportunity of verifying the occurrence of such cases. There is therefore reason to believe that these slow forms have neither been observed nor described for a very long time. Hence I deem that the record of a case that I met with last February in Rome will not be entirely without interest.

A young English officer in the Indian Army, endowed with a healthy physical constitution, but of lymphatic temperament, was sent, in the winter of 1890, from Calcutta to a garrison on the coast of Burmah, a marshy and unhealthy place. After remaining there for some time, he contracted intermittent malarial fever, which, though regularly treated, did not cease to return, varying also in type. In consequence of this, his strength being exhausted by the increasing paludal cachexia, it was recognized that in such a state of health the young officer was not able to continue his military service. Permission was therefore given him to return home. In the midst of his family, and surrounded by affectionate care, his general health began to improve, but the malarial fever, far from abating, definitely established itself in that type in which the fever recurs every fourteenth and fifteenth day. He and his parents were advised to go to the Continent. In the beginning of February I was asked to visit him. From the account given me by his parents and by the patient himself, and from my subsequent examination, I would declare the existence of tumor of the spleen, hepatic hyperæmia, and an anæmic condition from paludal cachexia. This examination took place the day after the first paroxysm of an attack, which occurred, he assured me, on the fourteenth day after the preceding one; and I was able to observe the second paroxysm, corresponding to the fifteenth day of interval. The feverish

attack, preceded by long and intense cold, began late in the afternoon and lasted about two hours, after which there was a high fever, which began to decrease after five or six hours, and disappeared after profuse perspiration. No especial symptom worthy of notice accompanied the attack with the exception of headache, and some pain in the left hypochondrium. The day after the second paroxysm the apyretic invalid felt some abatement of strength, which he regained in the following days by means of adequate nourishment and curative treatment. Such being the facts, I could easily make the diagnosis of the *quatuordecimanam* and *quindecimanam fever* observed by our fathers and described by Werlofio. Satisfied, then, that it was a question of malarial fever of a slow type in a cachectic individual, I thought it desirable in the long interval to put him under a tonico-reconstituent treatment, which succeeded admirably. Then, the day before the usual relapse, I prescribed the antipyretic, which I repeated on the following day (although the first attack failed) in order to avoid the second. Considering, then, that although the type had for some time been that of *quarto-deciman* and *quinto-deciman* fever, it still maintained the character of the quartan and double quartan fever which the patient had suffered, I determined to prescribe for its cure the anti-quartan mixture of Cotunnio, which I had long used for quartan fevers, and always with entire success. In this case, also, the result was entirely satisfactory. On February 28th and March 1st, days upon which, according to the calculated facts, the feverish attacks were due, they did not appear, and the patient, improving greatly in his general condition, left Rome about the middle of March. Later, notices from Florence assured me that for the second time the relapse had failed in consequence of having continued the curative treatment that I had recommended, and which I counselled the patient not to abandon for at least two months after the last attack.

Up to the present time it is not known how zymotic fevers transform themselves in various types, and all the studies of pathologists do not seem to me to have succeeded in determin-

ing the question. It is necessary, therefore, in such fevers to pay attention to the entity of miasmatic causes, and to the clinical verity; to view not only the principle of the specific infection, but likewise the variety of complications which can accompany it. It appears, then, that Cotunnio, taking into account all the circumstances which can be associated with *quartan* miasmatic fever, was persuaded of the necessity of strengthening the antipyretic action of cinchona by other tonic remedies after recognizing the fact that cinchona alone is insufficient to cure this type of malarial fevers. Now these being the clinical facts, it seems to me that Cotunnio's position is a true one. It will perhaps interest you, my honorable colleagues, if I remind you of the composition of the anti-quartan mixture of Cotunnio, which is given in the *Materiæ Medicæ Compendium* of the late illustrious Prof. Giacomo Folchi, in vol. i. p. 97, under the article "Zedoaria."[1] It is said there how Cotunnio referred, *ad aures*, to Prof. G. De Matthæis this method of curing quartan fever. De Matthæis having experimented with it successfully, it was afterward interesting to Folchi to put it in practice. The results obtained by the latter confirmed those of the Clinic of Rome. I owe it to the experiences of those my two illustrious masters, that I have learned to appreciate the therapeutic value of the Cotunnian mixture.

Mistura Anti-quartanaria Cotunnii: Corticis cinchonæ, subtil. pulv., gram. 48; radicis zedoariæ, s. p., hydrochloratis ammoniæ, āā gram. 4; camphoræ rosæ, gram. $1\frac{1}{2}$. Misce exactissime et divide in partes æquales.

To be taken during the apyrexia (Folchi).

For the cinchona I substitute "quinine sulphate." Then I divide the dose in ten or twelve parts, beginning the administration of the remedy only twenty-four hours before the anticipated attack. This method I have found succeed better.

In the cases which are of the nature of marshy cachexia I substitute the "hydrochloras ammoniæ martialis" in the same quantity.

[1] Wild ginger.

I have described this case not only with the view of making a contribution to the history of slow malarial fevers formerly described by our predecessors and no longer recorded by recent writers, but also to make known how, in the marshy regions of India, one contracts malarial fevers and suffers from morbid consequences which it seems to me have no parallel among those verified in other marshy countries. The present case, and the one previously reported of *orchitis malarica*, are sufficient examples.

Rome, 1891.

A CASE OF ACROMEGALY, AND ILLUSTRATIONS OF TWO ALLIED CONDITIONS.

By FREDERICK A. PACKARD, M.D.

[Read January 6, 1892.]

While the above title expresses, in a certain way, the small series of cases that I desire to report, some further explanation is necessary as to my reasons for grouping or rather contrasting them with each other. The first case is one, I take it, of acromegaly proper; the second is an example of the collection of symptoms and signs grouped together by Marie (*Revue de Médecine*, January, 1890) under the name of *osteo-arthropathie hypertrophiante pneumique*, while the third case of which mention will be made is an example of hypertrophy of the pituitary body without any of the changes seen in acromegaly.

The specimens from the latter case are grouped with the two living subjects from the fact that the pituitary body has been so frequently found to be hypertrophied in acromegaly, and that this case would go to prove, were any proof necessary, that the hypertrophy of the pituitary body is in no wise the cause of that disease, but is merely one of the many other changes found to be present in those cases that have reached the autopsy table.

The first case that I desire to report is that of Daniel G., white, aged forty-five years, whom I first saw in 1885 when I was resident in the University Hospital, in the ward of Dr. H. C. Wood.

He was admitted to the University Hospital in the spring of 1885, being at the time in a very somnolent condition. From his mother, who accompanied him, it was learned that he had one brother and two sisters, who were

healthy and without any trouble similar to that from which he suffered. His father died of apoplexy at the age of forty-five years. His mother has since died of pneumonia at the age of sixty-three years.

Until the age of twenty he worked in an ice-cream saloon; then on a farm out West for five years; and until ten years before his admission to the hospital (*i. e.*, until 1875), in a brass foundry. He has never had syphilis, and most careful questioning has failed to elicit any history that could possibly point to specific infection. He has used alcohol, but not to an excessive extent.

Ever since he can remember, he has had large hands and feet; but he admits that his face has increased somewhat in size. When fifteen years old he was "as tall as most men are at twenty-one," and had reached his present height at the former age.

In 1877 he had pain all through his body, supposed to be rheumatic, for which he was treated by pine-needle baths until an intense and exhausting diarrhœa set in and he became very much reduced. Since that time he has been unable to work, and has had gradually increasing weakness in his legs, and at times vague pains through his body. He has had three attacks of somnolence, the first lasting for three weeks, the second and third attacks lasting "for a long time." At the time of his admission to the hospital his mother stated that his brain-power had failed for several years, and that he was apathetic and very irritable, but had had no convulsions or delusions. His face since the onset of his illness was "much swollen."

On May 20, 1885, the following note was made: "Man of large frame, with very white skin. When aroused by being asked a question, he falls asleep again as soon as he has answered. He answers very slowly and deliberately, with a long pause before speech begins. The urine and feces are passed naturally. Patellar reflex absent and sensation in feet impaired. Can walk about ward by aid of a cane."

Before this note was made he had had (after his admission to the hospital) one attack of stupor that subsided; and at the time when the above note was made he was just entering upon another spell of somnolence. The urine at that time was negative. This somnolence increased until, by May 23, 1885, the note states, he failed to answer questions and lay with the eyes partly closed.

He was put upon potassium iodide upon his admission to the hospital, the drug being given in doses of thirty grains thrice daily until May 24th, when it was doubled in amount. On May 25, 1885, the eye-grounds were examined, and, according to the notes kept in Dr. Wood's note-book, the optic nerves were found to be atrophic and there was marked deficiency of capillaries.

On May 27th, inunctions of mercury oleate were added to the treatment with potassium iodide.

During the summer of 1885, he was given large doses of the iodide and

mercury, these being finally discontinued on account of increasing weakness, and a tonic and stimulant line of treatment adopted.

By September, 1885, his mental condition was worse; unconsciousness would appear and remain for days at a time. He gave evidences of suffering from headache at times, even during these periods of apparent unconsciousness. Before and after these attacks he could frequently be seen sitting on the side of the bed, fast asleep, with his fork raised toward his mouth, in which he had deposited a mouthful of food without having chewed it at all.

Upon September 15, 1885, the notes state that he was then in one of these spells, which had begun with vomiting; that he lay perfectly relaxed; could not be aroused by violent pinching or by exposing the eyes to light.

During October and November, 1885, he improved, and was able to walk to the dining-room and feed himself. He still complained of intense headache.

In December, 1885, Dr. Norris examined his eyes and reported that there was partial optic atrophy on both sides with hemianopsia, both temporal fields being lost.

Upon May 14, 1886, Dr. Reeves examined his nose, and reported that he had narrow nostrils, with hypertrophy of the mucous membrane on the lower turbinated bones, but no indication of pressure, and that the bony swelling on the outside of the nose did not extend inward.

Until his discharge from the hospital on November 13, 1886, he suffered from excruciating headaches that were not relieved by any treatment. He was given potassium iodide and mercury to the point of tolerance, a prolonged course of gold and sodium chloride, tonics, and stimulants; while, for his headache, potassium bromide, tincture of cactus, caffeine, theine, hyoscine, blisters, and the actual cautery were all tried, with but little or no benefit. I saw him again two days ago, and then made the following notes:

Since leaving the hospital in November, 1886, he has constantly had severe generalized headache with apparently causeless exacerbations, showing no periodicity. There is a constant feeling of weight in the vertex, and every now and then there is an agonizing feeling as though something were within the skull "scraping away the brain from the inside of the head from before backward." He has had no spells of somnolence since he was seen by me before; on the contrary, he sleeps but little. His general health has been fairly good, but his muscular strength very slight. Cold weather agrees with him better than warm, as during the summer he sweats a great deal and feels much oppressed.

He says that his memory is very poor, but he remembers readily persons that he saw and events that happened when in hospital seven years ago. He has had no convulsions. The senses of hearing, taste, and smell are normal. Vision is very poor, especially with the right eye, and he cannot see with that eye persons approaching from the right side.

For fifteen years he has had no venereal desire or power. His appetite is

poor, digestion good, no vomiting. His bowels are inclined to be costive. He gets short of breath readily and is tired by walking a few squares. His feet swell a little toward evening. He has no cough.

He is now five feet one and a half inches in height, and weighs 210 pounds. There is no sign of an excessive amount of adipose tissue. He talks very slowly and deliberately, and is rather lachrymose. His face is very large and heavy, certainly more so than in 1885–1886. The brain-case is relatively much too small. The forehead slopes strongly backward, the supra-orbital ridges being massive. The breadth of the brow is $5\frac{3}{8}$ inches; circumference of head $24\frac{1}{4}$ inches; biparietal diameter of head, $6\frac{3}{8}$ inches; occipito-frontal diameter, $8\frac{3}{8}$ inches. The ears are not disproportionately large. The eyes are relatively small when seen between the thick and heavy eyelids. The nose is very large and almost massive; the malar bones too heavy for the rest of the face. The chin is somewhat prominent, but not very markedly so. The skin of the face is very white, contrasting strongly with his thick black hair. The lips are large, thick, and of a deep-red color. The tongue is very large and thick, the mucous membrane thickened, and the surface much furrowed. The teeth are in bad condition, and are very small for the size of his mouth and jaws. The alveolar process of the lower jaw is considerably broadened.

Dr. G. E. de Schweinitz kindly examined the eyes and sent me the following report: "Refractive error myopia. V. in O. D. with correcting lens equals $\frac{6}{27}$; in O. S. with correcting lens $\frac{6}{9}$. There are rapid vertical nystagmus and divergent squint of the right eye, owing to paresis of the right internal rectus muscle. The double images are fused by a prism of 8°. The pupils are equal in size and react promptly to the changes of light and shade. Careful examination failed to reveal any hemiopic pupillary inaction. Each optic disc is oval, exceedingly gray, its centre containing a shallow cup. There is not much change in the size of the retinal vessels. The lack of capillarity and the atrophic pallor of the disc is most marked upon the right side. The field of vision shows marked contraction in all directions save toward the nasal side, but chiefly in the upper and outer quadrants."

The pharynx is capacious and there is a chronic catarrhal inflammation of its mucous membrane. The cartilages of the larynx do not seem to be abnormal. All of the cartilaginous portions of the nose are very thick. The large turbinated bones are plainly seen on but moderate separation of the alæ.

The thyroid gland cannot be felt. There is no dulness over the upper portion of the sternum. The circumference of the neck is 16 inches. The shoulders are sloping and inclined forward. The scapulæ are prominent, but not enlarged. There is a marked forward curve of the upper portion of the spinal column beginning at the fifth dorsal vertebra, while there is some lumbar lordosis with corresponding prominence of the abdomen and lower portion of the thorax. The spinous processes are in a straight line and are not enlarged.

The chest is very irregular in shape, some ribs being very large with numerous nodosities on their surfaces. At the junction of the manubrium with the body of the sternum there is a very marked prominence. The sternum measures nine inches in length. The xyphoid cartilage is not prominent. The second, fifth, and sixth ribs are *very* prominent, but all the ribs show roughness upon their surfaces. The junctions of the eighth ribs with their costal cartilages show very marked swellings, plainly visible and palpable. The costo-sternal angle is very oblique. There is no abnormality discoverable in lungs or heart. There is but little axillary or thoracic hairy development. The crests of the ilia are not notably enlarged. There is no abnormal change in the areas of hepatic or splenic dulness. The shoulder- and elbow-joints show nothing abnormal. The bones of the arms and forearms do not appear to be altered in any way; the muscles of these parts are, however, very soft and flaccid. The distance from tip of acromion to external condyle is $13\frac{1}{2}$ inches; from olecranon to styloid process of radius $11\frac{3}{4}$ inches.

The wrists are large but not deformed, measuring 8 inches in circumference, $1\frac{2}{3}$ inches in thickness.

The hands are seen to be very large, but not deformed. The fingers look stumpy and square at the tips. The skin of the hands is soft, pliable, and entirely hairless. The nails are stubby and longitudinally fissured. The thickness of the thenar portion of the hands measures $2\frac{1}{4}$ inches. The breadth of the hand at the level of and including the distal end of the metacarpal bone of the thumb is $5\frac{1}{8}$ inches; at the level of the heads of the metacarpal bones of fingers, but excluding that of the thumb, the measurement is $4\frac{1}{4}$ inches.

The length of the thumb is 3 inches, that of the middle finger $4\frac{1}{2}$ inches. The circumference of the last phalanx of the thumb is $3\frac{4}{10}$ inches, that of the middle phalanx of the middle finger $2\frac{4}{5}$ inches.

In the hands temperature, pain, and tactile senses are normal. Dynamometer registers in the right hand 97, in the left 65.

The biceps-jerk is present, but not marked.

Station is very swaying, partly no doubt from weakness.

The thighs and legs are small when compared with the large knees and feet. The length of the thigh from trochanter major to internal condyle is 17 inches, that of the leg measured from the upper edge of the patella to the external malleolus is $19\frac{1}{2}$ inches. The knees measure $16\frac{3}{4}$ inches in circumference, the greater portion being made up by the inner condyles and heads of tibiæ. The patellæ are large, but not out of proportion. Patellar tendon-reflex is absent on both sides, even with reinforcement.

The feet measure $11\frac{1}{2}$ inches in length, $4\frac{1}{4}$ in breadth at their widest part. The distance between the malleoli is $3\frac{1}{8}$ inches. The great toes measure 3 inches in length, $4\frac{1}{2}$ inches in circumference. Temperature, pain, and tactile senses are well preserved in lower extremities.

The urine is acid; specific gravity 1022; marked albumin-reaction with

Heller's and the trichloracetic acid tests, a slight deposit with picro-citric acid, and with heat a marked precipitate that cleared up with the development of a strong red color upon the addition of nitric acid; no sugar, casts, or crystalline deposit.

The second man is the example of *osteo-arthropathie hypertrophiante pneumique* of which I spoke.

He is a patient in the Episcopal Hospital, and it is through the kindness of Dr. D. J. M. Miller, who is now in charge of the medical wards there, that I am enabled to show him here.

He is single, aged twenty-nine years, and was formerly a carpenter. His father is living and healthy; his mother died at the age of fifty-seven years, of, possibly, phthisis. One paternal aunt died of consumption, as did also one maternal aunt. The rest of the family history is entirely negative.

At the age of fifteen years, while working on a farm, he began to have a slight hacking cough, and spat up some blood. The cough continued and he began to have night-sweats. At about this time he noticed that his finger-tips and nails were becoming rounded. At the age of twenty-four he had an attack of "dysentery," which lasted three months. He says that during this attack he had fifteen stools daily, without blood but with much straining. For a year after this attack he ceased coughing, but at the end of that time the cough again set in. During his attack of "dysentery" he is said to have had "hectic fever." At the age of twenty-eight years another attack of "dysentery" began, and has recurred off and on ever since, with, however, intervals of freedom. While the bowels are loose expectoration ceases, to begin again when the movements return to their normal number. At the close of this second attack of acute intestinal disturbance his weight had fallen to 108 pounds; but in three months came up to 167 pounds.

As has been said, his finger-tips began to become rounded at the age of fifteen. He states that at one time, about three years ago, he was able at will to produce "dislocation" of various joints, notably of the hips and knees. He has had frequent spells of rheumatoid pain in various joints, and at times his hands are so stiff that he cannot do more than semi-flex the fingers. For seven or eight years after his cough began he was stiff in all his joints whenever he started to move.

About one year ago he had a peculiar attack that came on suddenly and consisted in a feeling of numbness all down the right side, including the face, followed by falling toward the right side. This attack lasted for five minutes, leaving no prolonged sequelæ, and never being repeated save for a slight transient sense of numbness upon the right side of the face when he has held his head in certain positions for any length of time.

His back has been bowed from almost the onset of his chest trouble—the cough, clubbing of fingers, stiffness in joints, and arching of the back having all been first noticed at very nearly the same time.

He has never had syphilis, nor has he been addicted to the use of alcohol. He has never had more than a very small amount of sexual desire.

His memory has been failing for about five years. He never has headache. The senses of hearing, taste, and smell are normal. On examination he is seen to be much bowed. He stands with the head thrust forward between his high shoulders. His head is large, face rather small. The lower jaw is slightly prominent, the horizontal ramus measuring 4½ inches; the angle formed by the two rami being very oblique. The malar bones are somewhat prominent. The ears project sharply from the skull. The teeth are in good condition save that many have been extracted. The alveolar borders are normal. The tongue is clean. Laryngoscopic examination shows nothing abnormal. The eyes are natural in appearance, the pupils equal and reacting well to both light and accommodation. There is no gross change in the fields of vision. The eye-grounds show no pathological changes.

The thyroid gland cannot be detected by palpation. The thorax is markedly bowed forward at its upper portion, there being marked dorso-cervical kyphosis, of which the most prominent part is composed of the seventh dorsal vertebra. A plumb-line dropped from this point swings four inches clear of the sacrum. There is but slight development of hair upon the trunk.

The chest is symmetrical, but the right side moves poorly in respiration. The apex-beat is in the normal position and the area of cardiac dulness occupies the normal extent of surface. At the apex the heart-sounds are normal; at the pulmonary cartilage the second sound is accentuated, as it is also at the aortic cartilage.

Over the whole left pulmonary area there is markedly exaggerated resonance. On the right side anteriorly there is marked dulness with cracked-pot sound in the fourth interspace. Posteriorly also there is dulness over the whole left side, except for an area of hyper-resonance above the level of the seventh rib. The breath-sounds on the left side are somewhat puerile in character. Over the right side, as a whole, there are distant, faintly heard, tubular breath-sounds with cavernous breathing and whispering pectoriloquy anteriorly down to the fourth rib, and posteriorly down to the level of the seventh dorsal spine. Posteriorly over this area of cavernous breathing there is also metallic tinkling. Percussion with coins fails to give the bell-sound. The expectoration is profuse, muco-purulent, of fetid odor, and contains neither tubercle bacilli, elastic tissue, nor fatty acid crystals.

Over the upper portion of the sternum there is no area of dulness that can be separated from that of the right side of the thorax.

The scapulæ are normal, save that on each side there is a marked bony prominence on the inner side of the mid-point of the spinous process.

The shoulders are somewhat large, chiefly from uniform increase in the size of the peripheral extremities of the spinous processes of the scapulæ. There is no limitation in the motion of the shoulders. The upper arms are

small, but show no deformity. The forearms are also small, but toward the wrist-joints there is gradual swelling and broadening until the wrists are reached. This swelling is chiefly upon the radial side and is not very marked.

The elbow joint measures	24.5	cm.	in circumference.	
The wrist " "	18.75	cm.	"	
" " "	7	cm.	in breadth.	
" " "	4.25	cm.	in thickness,	

The hands show a peculiar deformity, almost, if not quite, confined to the last phalanges of the fingers and thumbs. This deformity is symmetrical, and the description of one will answer for both hands. The carpal and metacarpal portions present no abnormality. The fingers are natural until the last phalanges are reached, when there is seen to be an increase in all of the diameters, giving to the fingers the appearance, suggested by Marie, of drumsticks. The nails are large, strongly curved from side to side and from base to edge. These arcs formed by the nail above and by the pad of the finger below give the appearance of the recurved beak of a paroquet. The nails are somewhat waxy, livid, and brittle, one or two of them being longitudinally split. (The man states that he can readily split the nails by means of a pin.) The nails seem to be merely lying upon the surface of the skin, there being no bed, but the edges of the nails reach to or overlap the true skin surface of the fingers. There is no marked tendency to the production of sweat on the skin covering the dorsal surface of these phalanges, such as has been noted by Marie. This enlargement of the finger-tips seems to be made up of all the tissues, but chiefly by change in the bones. The skin is certainly rather thinner than that covering the other portions of the fingers, and there is no evident subcutaneous thickening or induration. The thumbs are affected equally with the fingers.

The following measurements were taken:

Middle finger from metacarpo-phalangeal joint to tip	11.5	cm.
Breadth of middle phalanx of middle finger	2	cm.
" last " " "	24	mm.
Thickness of middle phalanx " "	19	mm.
" last " " "	20	mm.
Arc of nail	2	cm.
Breadth of nail	23	mm.
" first phalanx of thumb	23	mm.
" last " "	28	mm.
Thickness of first " "	24	mm.
" last " "	24	mm.
Arc of nail	25	mm.
Breadth of nail	29	mm.
Breadth of hand at level of metacarpo-phalangeal joints and excluding thumb	9	cm.

The grasp is very weak. Dynamometer: in right hand 36, in left 41. Sensation of all forms in hands is unimpaired.

Station is normal with eyes open and closed. The patellar tendon-reflex is normal on both sides. No ankle clonus could be developed.

The feet and legs were so much swollen from œdema (said to have been present for eight years, but to subside on lying down) that no measurements were taken save at the ankles, where, after the serum was displaced, the distance from internal to external malleolus was found to measure 91 mm.; and at the knees, the circumference of which was 39 cm. when the patient was erect. The patellæ are not enlarged nor is there any bony deformity of the joints. The tibiæ seem large and heavy. The feet are large but chiefly at the last phalanges of the toes, where almost the same appearances are found as are present in the hands.

The urine contains no sugar; a moderate amount of albumin; no casts or crystalline deposit.

What I wish to say of the third case will occupy but a few minutes, as its interest lies solely in the pituitary body.

The specimen was found at the post-mortem examination of a woman under my charge at St. Clement's Hospital. She was a native of England, aged forty-three years, single. I found her in the ward when I went on duty in the spring of 1891, and obtained the following brief and possibly inaccurate history.

Nothing in the family history has any apparent bearing upon the case, nor does the history of her past life throw any light upon the etiology of her trouble. For thirteen years she has been blind, this blindness having gradually increased for a long but undeterminable period. For two or three years difficulty in walking had progressively increased until absolute inability was attained two months prior to her admission to the hospital. She complained much of intense pain in the extremities, chiefly nocturnal, with severe but intermitting cephalalgia. There was no trouble with bladder or rectum.

Examination showed that she was a small, spare, frail-looking woman. Face symmetrical, features small. Pupils equally and widely dilated, not responding to light. Both eyelids drooped and there was marked nystagmus, both probably due to blindness. Ophthalmoscopic examination showed marked pallor of the whole eye-ground, with a brilliantly white, sharply defined, contracted disc, and vessels reduced to minute threads. There was absolutely no reaction to brilliant illumination.

The hands and arms were not paretic.

She was absolutely unable to walk, but could feebly move the legs in bed. There was quite marked spasticity in the legs, but no contracture. On both sides knee-jerk was very much increased, ankle-clonus was easily obtainable, but no plantar reflex could be elicited. There was no nerve-trunk tenderness. Sensation in the legs was distinctly impaired.

A diagnosis of multiple syphilitic lesions of the central nervous system was made, and she was put upon ascending doses of potassium iodide with phenacetin *p. r. n.* for headache. She improved decidedly, although slowly, until March 10, 1891, when the right arm became weak and loss of sensation in the left arm was noticed. The iodide of potassium was then discontinued, inunctions of mercury being substituted. In ten days the gums were affected, but meanwhile the paralysis of the right arm diminished to such an extent that she was able to raise the hand to her mouth. This improvement lasted but a short time, when power again entirely disappeared in the right arm, and there was added paresis of the left side of the face. Coma then appeared and gradually deepened until she died. Convulsions did not occur.

Owing to lack of time and poor facilities, the brain, cord, and sciatic nerve alone were examined. The dura mater was slightly thickened. On removal of the brain there was left remaining in the sella Turcica a slate-colored globular mass, evidently an enlarged pituitary body. The pia over the convexity was normal; at the base it was thickened, opaque, and whitish in spots. This alteration was most marked in the interpeduncular space, while around the left facial nerve there was a dense white infiltration of the pia. The optic chiasm was flattened from above downward—the optic nerves being very small, while the optic tracts were nearly, if not quite, normal in size. Upon removing the mass in the sella Turcica, the latter was found to be much hollowed out and slightly roughened. The mass itself was globular, measured between a half-inch and three-quarters of an inch, and gave a cystic sensation to the touch.

After hardening in Müller's fluid, sections were cut, and the following conditions were found: There was practically complete atrophy and sclerosis of the optic nerves, sclerosis of the spinal cord in patches, but chiefly involving the lateral columns of the lumbar and dorsal regions; the mass lying in the sella Turcica proved to be composed of hypertrophied glandular portion of the pituitary body with a few cystic cavities, and some increase of fibrous tissue at the periphery.

As I said in the beginning of this paper, these cases are not reported as in any way related or as links in a chain, but are shown together because the first two illustrate two conditions that were at one time mistaken for each other; the specimen of hypertrophy of the pituitary body is shown not only because it is a rare lesion, but also because in many of the reported autopsies upon cases of acromegaly a similar condition has been found, and because a similar condition in the case of acromegaly shown here to-night would readily explain the hemianopsia previously present.

In regard to the second case shown, I would venture the opinion that the condition of the right thoracic cavity is due to an old empyema with thick walls that has at some time opened both into a bronchus and into some portion of the intestinal tract, producing the signs presented upon physical examination, the former attacks of diarrhœa, and the peculiar peripheral lesions which Marie is inclined to assign to a poison created in the lung and selecting the bony and articular portions of the body in a manner analogous to that shown by the rheumatic poison.

DISCUSSION.

Dr. F. X. Dercum: This man, Daniel G., was my patient a number of times. I distinctly remember his somnolent condition, which extended over a period of many months. Even while eating he would fall asleep between the bites and would have to be continually aroused. His headache was intense. He was treated with iodides and mercurials under the idea that the condition was specific. No impression was made upon the case, and later I came to the conclusion that it was a case of acromegaly. At this time I succeeded in obtaining the photographs which I show. These represent his appearance when twenty or twenty-two years of age, and are in marked contrast with his present appearance. This case is of interest, as so few cases have been seen in this country, and is especially interesting in connection with the fact that Dr. Hare has recently shown a patient presenting similar changes.

Dr. H. A. Hare: The patient shown to-night very closely resembles the picture in Souza-Leite's thesis on the table. My patient also closely resembles the picture in the book. She has profuse sweating in the hands and the peculiar eye symptoms. There is unilateral hemianopsia.

Dr. G. E. de Schweinitz: I will add a word in regard to the examination of the eyes made some years ago at the end of the long period of somnolence. I have here a copy of the fields of vision as they then existed, namely, typical bitemporal hemianopsia. An interesting point is the irregularity in the dividing line. It is slightly in advance of the fixing-point, leaving the macula within the area of vision; the dividing line inclines from the centre above and below. There is no record whether the hemiopic pupillary inaction was present or not; at that time Wernecke's symptom was not so closely investigated as it now is. Dr. Packard tells me that this inaction is now demonstrable. This, in connection with the bitemporal hemianopsia, locates the

lesion anterior to the primary optic centres. The hemianopsia is said to have lessened, and in this respect the case accords with others in which the blind area has diminished in size and even cleared up entirely. At the time my observations were made vision was about one-half of normal, and the patient tells me that he sees as well as he did at that time. The optic nerves were then partially atrophied. So far as I know, there was no antecedent neuritis. The original diagnosis was a syphilitic deposit pressing upon the optic chiasm.

Dr. Frank Woodbury: In connection with the cases reported to-night, I recall one which I saw some years ago, of true acromegaly, etymologically speaking, for the enlargement was limited to one finger. The patient was a laboring man, admitted to the Pennsylvania Hospital about fifteen years ago. The middle finger of one hand was enlarged in all directions, and the hypertrophy was attributed to traumatism. It was an inch longer than the normal, if I remember correctly. At all events the finger was amputated, and is still preserved in the Hospital Museum. The case was reported by Dr. J. A. Lippincott, in the *American Journal of the Medical Sciences* for 1876.

The cases of acromegaly reported to-night, being clearly a systemic affection, would more properly come under some more general designation, such as "maladie de Marie." We want a comprehensive term to indicate this general affection involving, as it does, various bones of the trunk as well as the fingers and toes, and other tissues, for which acromegaly seems a very poor designation.

The symptom of morbid somnolence, reported in the first case, is exceedingly interesting, as we know so little of the cause of this condition. Without considering traumatism, or gross lesions of the brain which may interfere with its functions, there is one large class of cases where the disease may be considered dynamic, and which is called hysterical, hystero-epileptical, or cataleptoid, and after the somnolence or trance is over, there is no apparent disturbance of the general health. A second form, in which the somnolence is constant although not so marked, is frequently seen accompanying indigestion, when the patient complains of constant drowsiness. Here the health of the body and intellectual vigor are both impaired. The notes of the first case recalled to my mind the "sleeping-sickness" of the natives of equatorial Africa, which, I believe, always terminates fatally after several months of stupor. I have had conversations upon this interesting subject with missionaries who have lived among the natives for many years, and, while the pathology of the disorder is not clear, I have always been under the impression that this was a dietetic disorder. This view has been confirmed in a measure by the Rev. Dr. R. H. Nassau, a graduate of the Medical Department of the University of Pennsylvania, who recently returned to this country after living thirty years in Africa. This gentleman told me that he had observed this peculiar condition, and attributed it to the use of cassava

as food. The variety of palm growing in this part of Africa, the *Jatropha manihot*, from which the cassava is obtained, is a poisonous variety, and to its toxic influence he attributes the sleeping-sickness. I had never heard this explanation before, and think that it is new and possibly true. By the natives the sleeping sickness is regarded as incurable; but if it be caused by a toxic agent, or even by a too restricted diet, it may be cured by rational measures directed toward removing the cause and counteracting its effects.

SPECIMEN SHOWING MENINGITIS PROBABLY DUE TO INFLUENZA.

By FRANK WOODBURY, M.D.

[Exhibited January 6, 1892.]

I have the honor to present a specimen that I think will be of interest. It was taken from the body of a man dying of meningitis probably resulting from the poison of influenza. On Monday, December 27th, about noon, this man was brought to the Medico-Chirurgical Hospital. He was actively delirious, and had to be restrained in bed. He moved his right arm and leg more than the left. The muscles of the right side of the face were also mobile, and the muscles around the right eye were in a constant state of agitation. The left side of the face, although moving occasionally, was comparatively placid. His temperature was 104°. Under the use of ten-grain doses of acetanilide repeated several times the temperature sank to 100°, but reached 103.5° the next morning, and remained high all day in spite of cold sponging and the ice-cap to the head; it was still high in the evening, 102.5°, and he died comatose early the next morning. During the time that he was in the hospital he did not regain consciousness, but continued delirious, talkative, and restless until coma set in and he died of exhaustion. He passed urine freely; it was not albuminous. Examination of the chest failed to detect any sign of pneumonia, or of cardiac disorder. The pupils were regular, about normal size, and sluggish in response to light. On testing the vision (Dec. 28th) I found that the left retina was quite insensitive to the approach of the finger, while on the right side there was appreciation of objects and frequent movement of the eyelids. He was quite deaf, and paid little or no attention to questions.

The diagnosis of meningitis, or an irritative lesion, involving principally the right hemisphere, occurring in the course of an attack of influenza, was made. The history previous to admission was that he had worked steadily at his trade of stone-cutter up to Christmas eve. He then came home and ate his supper, and retired to his bed about eight o'clock, as he did not feel well. He had not been indulging to excess in alcohol. For a day or two he

had complained of feeling poorly, and of pain in his back, and remained at home in the evenings. Later he complained of headache and had a severe chill, during which he vomited. The next day he had a severe headache, and he remained at home. He was not confined to his bed until Sunday, when Dr. A. E. Roussel saw him, and on Monday sent him to the hospital.

The family was adverse to an autopsy; but, remembering the case that occurred at the Philadelphia Hospital some years ago, and one recently reported by Dr. Estel, of Brooklyn, in each of which a piece of knife-blade was found penetrating the skull, but no history of the injury was given. I insisted upon an autopsy, to discover the cause of the irritative lesion and of the brain symptoms.

The autopsy was made eight hours after death. The body was that of a well-nourished man of twenty-four or twenty-five years. Rigor mortis was present. There was some congestion of the lower parts of the body, but no eruption and no petechiæ. Upon opening the brain-cavity the dura mater was found absolutely healthy. On taking out the brain some purulent fluid was left in the cranium. Along the convexity of the brain, on both sides of the median fissure, was a layer of yellowish, slightly green lymph lying under the arachnoid. The vessels of the pia mater were filled with dark blood, and in places on the surface they were very large. What particularly interested me, in connection with the blindness of the left eye, was a mass of enlarged vessels at the posterior part of the right hemisphere, over the region of the angular gyrus. This was also present, but less marked, upon the left side. At the base of the brain was a moderate amount of lymph around the large nerves. The same was found on the cerebellum. The interior of the brain seemed healthy. There was a little more fluid in the right lateral ventricle than in the left; no marked engorgement of the choroid plexus was noticed on either side.

This specimen may throw some light upon the cause of the headache and of some of the symptoms which characterize the nervous form of influenza. This might have been considered as a case of simple meningitis; but meningitis does not occur without a cause, and I think that here the exciting cause was the influenza-poison. I hold this opinion for two reasons: First, we have the positive evidence of the influenza prevailing as an epidemic; and, secondly, we have the negative evidence of the absence of any other preceding active cause, such as a blow upon the head, alcoholism, acute croupous pneumonia, or Bright's disease. He also had premonitory symptoms of lassitude, pain in the back, chill, and vomiting, which, without an autopsy, would certainly have warranted a diagnosis of influenza.

The specimen is of interest also from the fact that pneumonia is a common accompaniment of influenza, and from the well-known association between pneumonic inflammation and inflammation of the meninges, there is no reason why meningitis might not occur either as an accompaniment, or as an alternative, of inflammation of the lungs in influenza. Two varieties of bac-

teria have been found in the pneumonia accompanying influenza—the micrococcus of Pasteur and the well-known diplococcus of Fraenkel—both associated with the streptococcus pyogenes, which is considered the constant bacterial form, and, by Ribbert, has been declared to be the cause of influenza.

It is of further interest in another direction, and that is the possibility which has been broached that the epidemic of influenza may be followed by an epidemic of cerebro-spinal fever. In the previous epidemic of cerebro-spinal fever in 1873, I made autopsies upon a number of cases, and the lesions of the brain, as I recall them, were identical with those found in this specimen. This, however, was not a case of cerebro-spinal fever. We have no epidemic of cerebro-spinal fever in this city at present, and the patient presented neither petechial spots upon the surface, nor other symptom of this fearful affection. The other explanation seems to fulfil all the requirements of diagnosis, and I have therefore considered this to be a case of meningitis due to influenza.

MORPHŒA WITH MACULÆ ATROPHICÆ.

By LOUIS A. DUHRING, M.D.,
PROFESSOR OF SKIN DISEASES IN THE UNIVERSITY OF PENNSYLVANIA.

[Read February 3, 1892.]

The patient is an Englishwoman, fifty-five years of age, a brunette, spare, but in good general health. There is no apparent cause for the disease. It first manifested itself a year and a half ago, and has been gradually spreading. The regions invaded are the nape of the neck, and the adjoining scalp to a slight extent; the chest just below the left clavicle; the wrists and forearms. Several stages of the process exist, and the lesions are so different as to require separate description. Three distinct kinds are noted:

1st. Whitish patches of skin with manifest structural change in the true skin, of the nature of a peculiar fatty degeneration of this structure, constituting the commonest form of the disease. 2d. Distinctly circumscribed, depressed, and cicatriform whitish spots, varying in size from a small to a large pea, which plainly exhibit wasting and thinning of the true skin, the lesions resembling scars from syphilis, being thin, soft, and pliable. 3d. Patches of mottled, brownish-red, pigmented, structurally altered, atrophic skin, with a broken border or margin of firm, variously sized, irregularly shaped, papular elevations. Over these patches here and there are distinctly marked bluish-purplish veins running in various directions. The skin in the central portion is thinned, and the border, as stated, is thickened, but nowhere is the skin bound down to the subcutaneous tissue, the whole patch being freely movable

over the tendons and fasciæ. 4th. Enlarged, bluish-purple veins, identical with those on the patches of the wrists, which run up the forearm and are not associated with the other forms of disease described, although they are in the neighborhood of some atrophic macules. Having thus outlined the chief features which characterize the affection as a whole, the individual lesions and their distribution upon the several regions involved may be referred to more definitely.

On the back of the neck at the line of the hair, partly on the scalp and partly on the non-hairy portion of the neck, there exists an irregularly shaped, sharply defined, whitish patch of skin, the normal structure being changed into a whitish, lardaceous, non-indurated, soft, pliable, freely movable patch. The sense of touch with the fingers does not determine any increased thickness or structural change, so that in picking up the skin with closed eyes one would hardly detect disease. This patch is about two inches in diameter and is not surrounded by any border or hyperæmic zone or by injected veins. The hair of the scalp growing from the patch is of natural color, blackish, and not whitish as might be expected. This lesion represents one of the varieties of morphœa, and according to my experience the earliest, simplest, and mildest phase of the disease.

Near by this patch, on the back of the neck are several pea-sized, rounded, sharply defined, slightly depressed, shallow, whitish or pearl-colored, atrophic macules, with thin skin which at first glance resembles scars from the large pustular syphiloderm or from burns resulting from the application of a hot iron. They are disseminated upon the neck on either side of the median line, and show no special distribution or arrangement. They are in no way different from the typical maculæ atrophicæ which are met with occasionally upon various regions, and usually without other forms of cutaneous disease.

Upon the flexor surfaces of the wrists are two symmetrical, rounded, atrophic patches, the size of a silver half-dollar, defined in outline with a raised, indurated, irregular, uneven

papular border, and pigmented, of a mottled, brownish-red hue. The central portions of these patches are somewhat wasted and depressed, the skin being thinned, but soft and supple. Some enlarged and purplish veins run irregularly over the surface. On the flexor surface of one forearm, running up toward the elbow, there exist several pea-sized, whitish, atrophic macules, identical in character with those on the back of the neck. They incline somewhat to take on a linear arrangement rather than to be widely disseminated.

The lesions which have been described constitute the whole disease. The several varieties have no association with one another, but they are plainly due to the same cause. They are not stages of one process, but are distinct forms of cutaneous change, beginning and running their course as such. The patient complains of no pain or serious inconvenience from the disease, and seeks medical advice because of the disfigurement and of the tendency of the process to waste and atrophy.

The case represents an unusual phase of the somewhat rare disease morphœa. Many years ago I pointed out that this affection was characterized not only by the so-called "patch," but moreover in some cases by a variety of lesions, which might occur either singly or in combination. In the third edition of my *Treatise on Skin Diseases* (published some years ago), attention was directed to the observation that atrophic macules sometimes were present with the characteristic lardaceous patch. The occurrence of these two forms of lesion together, however, as in the case before us this evening, I regard as rare, one or the other variety of atrophy usually existing alone.

Morphœa must be classified with the atrophies, and not with the hypertrophies, as some prominent authors have done. The process is distinctly atrophic in all its essential features, especially in its course and termination, which are characterized by degeneration of the skin and subcutaneous tissue with usually more or less thinning, shrinking, and wasting or degenerative atrophy. The present case illustrates the close relationship

pathologically of maculæ et striæ atrophicæ with the common plaque of morphœa, as this latter was originally described by Addison and E. Wilson. At that date the affection seemed to have been scarcely known in Germany, and moreover even now it appears to be rarer there than in England or in this country. Many years ago, during a long sojourn in Vienna, with daily attendance upon the clinics for skin diseases, I do not recall having observed a single case.

Concerning the diagnosis, no difficulty can exist, it seems to me, if we are in the habit of studying cutaneous disease from the standpoint of anatomy and pathology, and more particularly the latter. This classification of skin diseases, upon the basis of general pathology, is not only the most scientific, but what is of more importance, is also the most practical and useful for our daily dealings with these diseases. The affection before us belongs manifestly to the atrophies, the process at work being essentially degenerative and atrophic in all its phases. This point established in our minds, there remains merely to find a place for it in this class, and it plainly must be grouped with atrophies of the true skin. Such forms of atrophy are comparatively rare, the true skin not being prone to take on atrophy as a primary process. The several affections of this kind which may be classed together are atrophy of the skin proper (atrophia cutis propria); maculæ et striæ atrophicæ; morphœa; and some forms of scleroderma, the two latter affections sometimes coexisting.

The treatment of these cases is generally unsatisfactory, the prognosis, however, depending a good deal on the variety of the disease present, and on the stage of the process. In some cases arsenic internally is useful, but in the patient before us local inunctions with stimulating ointments and oils, with massage, electricity, and frictions, will probably prove more beneficial.

DISCUSSION.

Dr. Francis X. Dercum: I would ask whether or not there is any modification of sensation in this case? Also, whether cases of this affection have been observed sufficiently long to determine whether or not the atrophy is limited to the skin, or whether the subdermal tissues are also affected?

Dr. L. A. Duhring: The subject of morphœa is an extensive one. My object was not to write a paper upon this disease, but simply to bring before the College a case with the clinical notes pertaining to that case. I would, however, state that at times there is want of sensation in the affected patches, while at other times there is no interference with sensation, notwithstanding there is considerable shrinking and change in the tissues. The disease often goes on to distinct atrophy. I have seen limbs so shrunken that the patient was unable to walk. It may affect other parts, as the elbow, or the mouth. These are, however, extreme cases, and have been reported under different names, sometimes as scleroderma. The two diseases should be differentiated, notwithstanding the fact that they sometimes coexist. Some ten or fifteen years ago I observed about fifteen or twenty cases of morphœa—a large number. During the past five years I have seen comparatively few cases. The chief interest in the present case is that it illustrates two diseases hitherto regarded as distinct, but which, I believe, should be considered as being closely connected. In this case they are due to the same cause, and have the same pathology, although showing somewhat different lesions.

Dr. A. van Harlingen: The case which Dr. Duhring has presented illustrates a large and increasing group of diseases of the skin dependent upon disturbance of the nervous system. The subdivision of this group to which the present case belongs is included under the title of trophic disorders. Morphœa is one of a series which begins with atrophy of the epithelium and pigment of the skin as seen in vitiligo, while at the other end of the series we find such conditions as hemiatrophia facialis, where the atrophy affects all of the tissues. Morphœa occupies a middle place in this series. I regret that Dr. Duhring has not gone more fully into the discussion of this subject, for I know that he is one of the few observers who have had an opportunity of making a microscopic study of the lesions. I have read an admirable account of the minute anatomy of this condition by Dr. Crocker, of London, in which reference is made to the publications of Dr. Duhring. If I understand Dr. Crocker, he states that the disease begins as a faulty innervation, and that there then results a small cell proliferation around the capillaries, which in time chokes the capillaries and prevents nutrition. The cells become lengthened, and a certain amount of connective-tissue formation takes place. This would account for what we often see—that is, a round patch changing into a brown, knotted scar. In the pathological collection of the University of Pennsylvania there is a magnificent wax model emanating from Addison, of London, and illustrating a paper on the disease known

as "Addison's keloid," in which the first of the series represents such a lesion as is seen here, and later the extreme degree of deformity to which Dr. Duhring has alluded. The disease is unquestionably due to some affection of the nervous system, and often follows the course of a nerve. Mr. Hutchinson has recently reported a number of cases, in many of which the lesion followed the course of the nerves. He even makes a sub-class of "trigeminal morphœa." I have myself under treatment at present a case in which the morphœa has followed the supra-orbital branch of the trigeminal nerve, beginning at a small patch on the inside of the nose and running up as an ivory-like, narrow band nearly to the vertex, one-third of an inch in width. That alone would show that the disease is connected with some trouble in the nerve. It would be of great advantage if we could have these cases carefully investigated, for it is only by studying the condition of the nerve-trunks that we shall gain any knowledge of the causation of these diseases. The atrophic maculæ, and the dropping off of fingers which sometimes occurs in leprosy, are probably connected with the same sort of a change in the nerve as causes the morphœic patches. If these cases were studied from this point of view they would be of great value. The study of neurotic diseases of the skin has not been pursued sufficiently to enable us to get at the bottom of the matter. In monographs on this subject allusion is made to the work of Drs. Mitchell, Moorhouse, and Keen as laying the foundation for these investigations; but since these admirable investigations little or nothing has been done to throw light upon the influence of the nerves on the nutrition of the skin. In dermatitis herpetiformis—that curious affection of the skin which, in compliment to the admirable clinical work of a Fellow of this College, is known abroad and in this country as "Duhring's disease"—and in herpes zoster, disease of the nerve-trunks has been found.

The milder forms of vitiligo and of linear and macular atrophy seem to be amenable to treatment. Vitiligo has been cured by nutritive medicine, and occasionally by the use of electricity, and I have no doubt that in time the other affections of this series will be found amenable to treatment.

Dr. Dercum: The remarks of Drs. Duhring and Van Harlingen have been interesting to me, and have thrown light upon these atrophies and those which I as a neurologist see occasionally, namely, hemi-facial atrophies. With regard to hemi-facial atrophy, it is interesting to recall the fact that in a post-mortem made in a case by Dr. Mendel, of Berlin, two or three years ago, he found degeneration of the descending root of the trifacial nerve, showing that the trophic fibres were contained in this root. The case was one in which the distribution of all three branches of the trifacial was concerned. The skin, subcutaneous tissues, and even the bones and muscles, were involved. The patient had died of an intercurrent disease—phthisis.

Dr. S. Weir Mitchell: What we want in these cases is a differentiation of the nerve lesions in relation to the changes which follow. These reports simply tell us that there were atrophic changes in the nerve, but the changes in the skin are varied. As it is, we are yet far from any rational pathology.

CYSTIC DEGENERATION OF THE MUSCULAR FIBRES OF THE HEART.

A Form of Disease Hitherto Undescribed.

By ARTHUR V. MEIGS, M.D.,
PHYSICIAN TO THE PENNSYLVANIA AND CHILDREN'S HOSPITALS.

[Read February 3, 1892.]

In April, 1891, I read before the College a paper entitled, "The Microscopical Anatomy of the Human Heart," which appeared in the Transactions of the College of Physicians of Philadelphia, and was published besides in *The American Journal of the Medical Sciences* for June, 1891. It was then shown that in the human heart in the natural condition the muscular fibres are penetrated by capillaries, and that they are not therefore, as is commonly supposed, mere solid rods. At the same time, I alluded to the fact that I was able to make this observation owing to a certain pathological change that I had noticed in studying diseased hearts—a change by which the nearly solid normal fibres became tubes. So far as my observations extend, this condition is usually most marked in the fibres of the papillary muscles of the left ventricle, though it is common in all other parts of the heart as well. The degree of the excavation varies exceedingly; the cavities may be so small that in some instances it is impossible to distinguish them from capillaries, or, on the other hand, the hollowing-out process may have gone so far that the fibres are changed into tubes with thin walls. The disease may be best studied when the fibres are seen in cross-section, for then its most character-

istic appearances are presented. Its presence can, however, be equally positively determined in longitudinal sections, though greater care and discrimination must then be exercised to recognize it. The drawing (Fig. 1) represents types of the morbid changes as they appear when present in a high degree of development. There are two fibres which are natural; the others have been more or less eaten away in their centres by disease. The destructive process, in its most extreme form of development, removes the whole of the muscular substance from the centre of the fibre, no part of which, when examined with the microscope, will present the usual appearance of muscular tissue except the thin outer walls, and even these (Fig. 1, No. 7) may show only in places the cross-markings characteristic of heart muscle. A curious feature is the way in which the muscle nuclei often lie loosely in the cavities, without attachment to the remaining tissue. This is very different from the natural condition when the nuclei are closely surrounded by the muscular tissue. This separation of the nucleus was very marked in the two fibres represented in Fig. 1 as Nos. 5 and 7. Nos. 3 and 5, and, in a slightly different stage, No. 4, represent another phase of the destructive process. In them, instead of a single large cavity in the centre, there are several smaller holes irregularly distributed through the fibre, and in No. 5 the muscle nucleus lies in the largest of these. The degenerative process is more advanced in No. 4 than in the two others, for in it the only muscular tissue remaining is that constituting the thin outer walls, with a little more which is irregularly distributed through the inner portions, and some shreds of a material which must be the endomysium or fine connective-tissue substance which normally exists in the muscular fibres. It must be understood that these cavities do not usually give the impression that during life they were empty, or even that they contained only a clear liquid, for there is always present more or less material that has no distinguishable structure. Areas presenting this condition are represented in the drawings by the dotted portions. It seems to me that this hollowing-out, when most extreme, is apt to be near the ends of the

fibres, toward their point of origin or of insertion. In sections of tissue that show it I have found the hollow fibres most numerous not far from the endocardium or pericardium, and less so deeply in the centre, though, as already stated, it will be unmistakably present in all parts of the heart. The heart which furnished the fibres represented in Fig. 1 was very fibroid, but the hollow fibres were not found in the fibroid portions of it. Fig. 2 was made by the photographic process alone, without retouching, and shows fibres in varying stages of degeneration.[1] In it may be seen several of the same fibres as are shown in Fig. 1. In this heart, however, I was able to study the disease to the best advantage because it was present in its most extreme form.

The patient was a man fifty-seven years of age, who had been very dissipated, having eaten and drunk too much, and having done most of the things a man should not do. He denied ever having had syphilis, but at sixteen years of age he had an attack of inflammatory rheumatism. He said he had been healthy until recently (or about six months before his death), when he became exceedingly short of breath, and had some irregular rheumatism. His condition became gradually worse until he died, after having suffered to an unusually great degree with difficulty of breathing. For many weeks there was general œdema and ascites, also albumin and casts in the urine, and great atheromatous stiffening of the arteries. The post-mortem examination showed fibrosis of most of the organs, including the heart, which was much enlarged. The kidneys were contracted, and there was an extraordinary amount of chalky deposit, which was not confined to the walls of the arteries alone, for there were large deposits in the mesentery and in the posterior walls of the abdominal cavity.

The first case in which I discovered the presence of the morbid change in the heart-fibres was that of a man past middle life, who had died with aortic regurgitation. He denied having had rheumatism and syphilis, and died of exhaustion after an illness of a few months, without ever having had dropsy, or dyspnœa, or any evidence of kidney disease. There was aortic disease, and the heart was enlarged. The kidneys were rather larger than normal, and but slightly diseased. Excessive hollowing out of the muscular fibres of the heart was found.

[1] The photograph, which is a very beautiful one, was taken by Dr. George A. Piersol, Professor of Anatomy in the University of Pennsylvania.

It is impossible at present to predict from clinical manifestations which cases will present this curious change in the heart. Some idea, however, of its clinical and pathological accompaniments may be gained by contrasting cases in which the disease was present, with others not so diseased. As was stated in my paper showing the existence of capillaries in the heart's muscle-fibres, I have in my collection sections of forty-nine human hearts, accompanied in most instances by clinical histories of the patients. In some typical cases of Bright's disease, both of the interstitial (or contracting) and of the parenchymatous forms, hollow fibres were present, and in other like cases I have not found them. In hearts showing great increase of fibrous material, it has in some instances been present and in others absent. It is a curious feature that the vacuolations, when present in a fibroid heart, occupy almost exclusively those portions of the organ not affected by the fibroid overgrowth; in such regions it will be conspicuous by its absence. There are in my collection sections from two hearts showing more marked fibroid overgrowth than any of the others; in one, the hollowing-out of the muscular fibres is very great, in the other there is none of it; in both cases the kidneys were very much contracted. The history of one of these cases has been given in some detail above. Examination of such sections as are in my possession shows the vacuolation of the fibres to be absent in cases of brain syphilis, sarcoma, general miliary tuberculosis, Bright's disease, pulmonary phthisis, typhoid fever, pneumonia, dysentery, epithelioma of the bowel, and aneurism, and to be present in what clinically was recognized as organic heart disease, Bright's disease, typhoid fever, ulcerative endocarditis, and in young infants that had died of wasting. I have sections from the hearts of a number of foundlings who died during the earlier months of life from the wasting so common among infants of that class, and in some instances vacuolation of the heart's muscle-fibres was unmistakably present, and in others absent. In many respects the histological condition of heart muscle in young infants, as might be expected, is very different from what it is later in life. In a child ten years of age who died with

dropsy, and had amyloid disease of the liver, spleen, and kidneys, and of the heart and lungs too, there was most extreme hollowing-out of the muscle-fibres. Some of the fibres in this case, when seen in longitudinal section, presented small bulbous-looking swellings at points where they were hollow, and thus making it appear that some distending process had occurred where the vacuolations existed. In another child of twelve, with cardiac hypertrophy and dropsy, the vacuolations were also present, but in much less degree than in the previously mentioned case.

It has been my endeavor to describe this pathological change so that it may be recognized by others; and, as previously stated, it was while studying it that I was led to the discovery that capillaries normally penetrate to the very centres of the muscular fibres of the heart. The kind of cases in which the disease was found I have also denoted. It is now desirable to understand, if possible, the nature and origin of the morbid process. The fact that the muscular fibres of the heart are penetrated by capillaries, and are not, therefore, truly solid bodies, together with the appearance of the spaces already described, lead to the almost inevitable conclusion that the process is one of cystic degeneration. The only other conceivable explanation is that the cavities are minute aneurisms, being dilatations of the capillaries after they have passed into the muscular fibres. Such an assumption, however, would seem to be negatived by the nature of the material which lies within the cavities. Any material visible is amorphous and granular, or is yellowish pigment in irregularly shaped flakes, all looking as if suspended in a liquid, and thus presenting the precise characteristics of any section of a cyst. If the process be one of multiple capillary aneurism, fresh blood should be found in the spaces instead of the detritus described. Such being the case, it would seem hard to escape from the conclusion that the cavities are true retention cysts, and that they are produced in a manner parallel to that of renal cysts. A capillary must become blocked in two places and the portion between these dilate, nad thus a cyst be formed. It is, of course, quite possible that the

vacuolations are false cysts formed by the escape of blood into the substance of the fibres (hæmatocele), or by degeneration and softening of the muscle-substance itself. This, though quite admissible as a possible explanation of the pathological process, is much less likely to be the real one than is the supposition that they are true retention cysts; for, in the first place, there are in the fibres cavities, the capillaries, in which cysts might form, and, in the second, the contents are precisely similar to those common to cysts. These cavities present another characteristic which is common also in renal cysts. Shreds, or festoons, or shelf-like projections of connective tissue hang irregularly across the cavities. Nos. 3 and 4, Fig. 1, exhibit this appearance.

If time and the progress of events should establish that these minute cavities in the heart's muscle-fibres are true retention cysts, having their origin within capillaries in the fibres, it will be the first time that it has been shown that cysts do originate within the vascular channels, as they are already so well known to originate within other channels, as, for instance, the renal tubules and other gland ducts. There seems to be no reason in the nature of things why such should not be the case.

The presence of capillaries within the muscular fibres of the heart, a fact in normal anatomy, and the occurrence of cysts within the fibres, a fact in pathological anatomy, and the fact that the one observation led to the other, are an illustration of the correctness of the view so much dwelt upon now, that the two branches of science, normal and pathological histology, have close and important relations. In this instance the train of events was as follows: First, as the result of pathological study, the recognition of the comparatively large cavities in the fibres, which at that stage were inexplicable. From this came the desire to examine more minutely the histology of the human heart, and this resulted in the observation that the capillaries not only closely surround the fibres, but that they actually penetrate them, a fact which seemed at first in no way connected with the holes eaten into the fibres by disease. Lastly, further

and more careful consideration leads to the conclusion that the larger spaces which result from disease are but cystic dilatations of the natural blood channels.

It is important to remember that this disease, which I have called cystic degeneration, is very common, and has been found by me to be present in many ordinary well-recognized diseases. What bearing it may be found to have clinically is, of course, impossible now to predict, but it seems not unlikely that it may be important. The thoughts certainly are new ones, both that cystic disease may have its origin in the vascular system, and that it is a common cause of degeneration of the heart, an organ which is so prone to degenerate, and degeneration of which is so far-reaching in its results.

Note.—It should have been mentioned that the sections of tissue from the appearances of which my conclusions were drawn were prepared as far as possible, upon a uniform plan, both for the preservation of the tissue and the mounting of the sections. I thought that if this was done the unnatural appearances could with more certainty be attributed to disease than if different methods were used, in which case it might be thought that what I described as disease was due to faulty technique. The tissues were almost all preserved in seventy per cent. alcohol, and the imbedding material was paraffine, except in a few instances, when celloidin was used. The staining material was borax carmine. Considering the imbedding material employed, and that the sections were often cut from parts of tissue far from the surface of the pieces, it would be unreasonable to suppose that what I have called hollow muscle-fibres were fibres from which the centres had fallen out in process of preparation, a possibility which would strike every practical microscopist.

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Fig. 1.

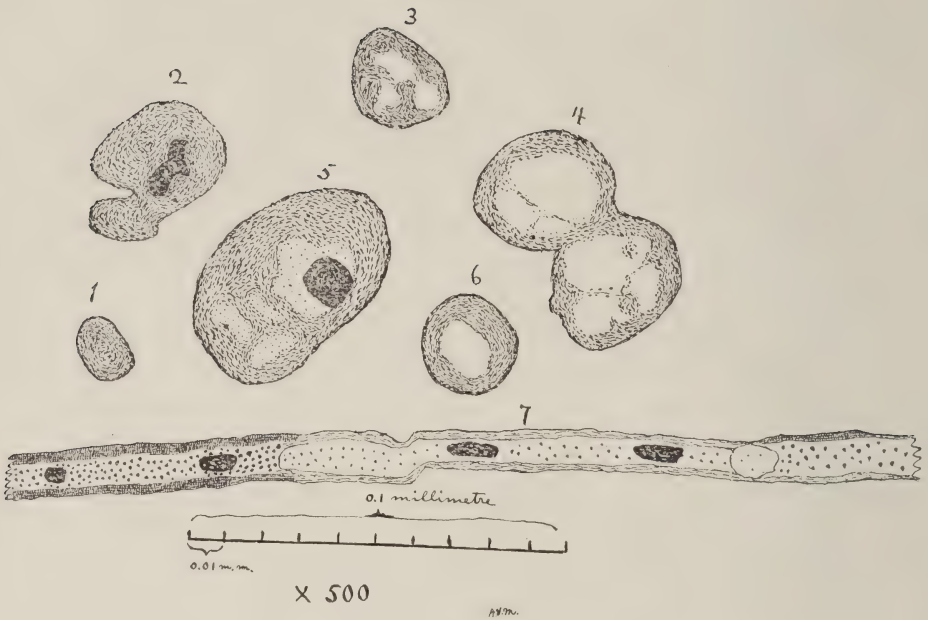
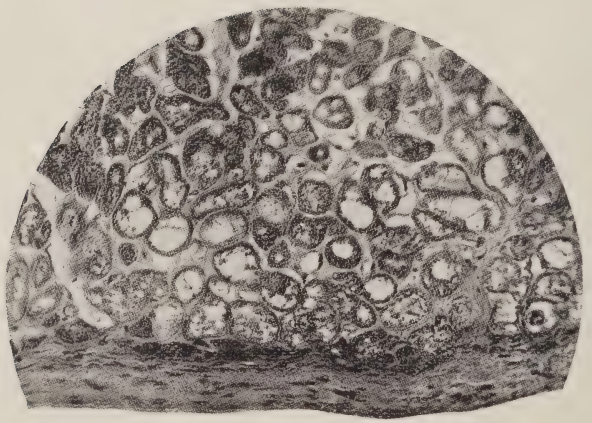


Fig. 2.



Explanation of Figures.

Fig. 1.—The Nos. from 1 to 6 inclusive represent heart muscle-fibres in cross-section, and were all drawn from the same microscopical preparation. No. 7 was drawn from a preparation in which the fibres were cut longitudinally. The camera was used, and they were drawn to scale, the amplification being five hundred diameters. Nos. 1 and 2 are ordinary fibres, and present no evidence of disease; they were put in the picture to contrast with the other fibres, which are diseased. The irregular shape of the nucleus shown in No. 2 is not unusual. The other fibres present evidences of advanced disease. Nos, 3, 4 and 6 look as if irregularly hollowed out, the dark areas being intended to represent the muscular tissue and endomysium (intramuscular connective tissue), while the lighter portions are areas from which the muscle-substance has disappeared as a result of disease. No. 5 is a large fibre, showing several spaces from which the muscle-tissue has gone, the space being empty, or filled with a more or less granular material. There is also a nucleus which lies in a space which is empty or filled with slightly granular matter, this being represented in the drawing by a dotted area. No. 7 is a drawing of a fibre cut longitudinally, and very well shows the irregular hollowing-out process or vacuolation which has taken place. The fibre looks as if it had been converted into a hollow cylinder divided into several compartments. The drawing was made to show that at the sides of the fibre the muscular tissue still persists, in places the cross striæ even being distinguishable, while the dots in the central portion are intended to indicate the more or less granular appearance. The nuclei lie as if they had been loose in the central space, not being in any part in contact with the muscular tissue remaining. The drawings are diagrammatic, but the dimensions and outlines are correct.

Fig. 2.—This is a photograph (a half-tone) of a single field under the microscope of the papillary muscle of the left ventricle from the same case as that from which the drawings were made. Several of the fibres in the drawing are in this picture. It admirably shows the manner in which the interior of the fibres has been irregularly eaten away by disease. Neither the negative nor the print from which this was made was retouched.

THE USES OF FEVER HEAT; THE OCCASIONAL DANGERS OF ANTIPYRETICS IN TYPHOID FEVER.

By J. H. MUSSER, M.D.

[Read February 3, 1892.]

Animal heat serves a physiological purpose in the economy When the degree of heat is beyond the limits of the norm its effects are not physiological. In health normal heat is a stimulus essential to the carrying on of physiological processes. Without it the nerve-centres controlling respiration, circulation, and other processes are dormant. No stretching of the imagination is necessary to conceive of such an impairment of the vital forces that increased heat or pyrexia may be essential as a stimulus to enfeebled nerve-centres.

The following observation impressed the writer with the usefulness of fever as a vital stimulant.

The patient, two years old, was ill of whooping-cough and its graver pulmonary complications—capillary bronchitis and atelectasis. Exhaustion was extreme on account of her long illness of three months. Fever was marked. During the last week of her illness death was daily, then hourly expected. The hands and feet became cold, the face pale, the respirations excessively rapid and feeble (100 to 110), the pulse feeble and irregular and so rapid it could scarcely be counted; a cold sweat bathed the brow, while stupor was present. These grave symptoms were first observed in the mornings. The temperature would be 100° to 101° F. at this time. As the day advanced the usual afternoon exacerbation of fever took place. With such degree of heat came evidences of renewed strength. The extremities warmed, the color brightened, the respirations were less labored, the pulse became perceptibly stronger and fuller and more regular; intelligence was restored; food, though refused before, was now taken with avidity, and even

asked for, and notice taken of objects and events. Hope was restored, to fall again as the thermometer went down.

At first these paroxysms of heat-exacerbation and stimulation took place daily. In the last three days of the child's illness, they took place every two or three hours. Indeed, during the period of lowered temperature, so depressed and feeble was the child that momentarily death was looked for. As the temperature rose—but not until it was above normal for some time—the grave symptoms disappeared. These hourly heat-paroxysms were closely watched, and to no other cause than the stimulus of heat could the revivals of strength and increased functional activity of the circulatory and respiratory apparatus be attributed. It is difficult to picture, and was certainly a most striking exhibition of the play of heat-centres in the phenomena of life. Finally after the stage of exhaustion they had undergone, the heat-centres did not respond to the unknown stimuli, the temperature did not rise, and gradually all function ceased.

I wished more particularly to place this observation on record in order that in these times when so much is said about the use of antipyretics, the dangers of fever, and the necessity of reducing temperature, we should bear in mind that high temperature may have a physiological use in the economy at certain times. I am well aware that certain German and French writers have held that high temperature is a conservative process, particularly in septic conditions through its effect upon the bacteria or the poisons generated by them. But, independent of this action, fever serves quite a physiological use in the economy. This is the only observation that I wish to record in regard to the usefulness of fever.

The following remarks are addressed to practitioners who continue to use, and think they must use, antipyretic medicines in typhoid fever because the thermometer records high temperatures. I have never used such means to reduce temperature in this disease, but I wish to particularly impress upon those who will use antifebrile drugs that it is essential they should hold their hands at least at times. The present intemperance in the use of antipyretics is due to laudation of this class of drugs by eminent therapeutists in days gone by.

Now in regard to the occasional danger of the use of antipyretics in certain cases of typhoid fever. There are three periods in the course of typhoid fever when, without doubt,

the use of antipyretics is dangerous, and these must be carefully considered. First, in the early or middle period of cases which come to us after removal from a distance. Sir William Jenner was the first to lay great stress upon the danger of the removal of a patient suffering with typhoid fever. Anyone who observes cases in the medical ward of a hospital will notice that the temperature for the first twenty-four hours after admission is unusually high—that is, higher than one would expect at the period that the disease has reached. This rise of temperature is undoubtedly due to the exhaustion that has taken place on account of the removal to the institution. I have seen this so often and made so many observations in connection with it that I hesitate to use any antipyretic during the first twenty-four hours after admission. The records which I have bear out the correctness of this course. Even if no antipyretic is given, the temperature usually falls to the proper point in twenty-four hours. Sometimes stimulants are required, and under their use the temperature falls. On the other hand, I have seen cases which had been admitted with a temperature of 105° or 105.5°, to whom an antipyretic was given by the resident with serious results. I am so sure of the inadvisability of administering an antipyretic under these circumstances that it is a standing instruction in my wards that no antipyretic be given during the first twenty-four hours after admission; but that stimulants shall be administered.

Again, there are certain cases in which peculiar idiosyncrasies exist, where it is inadmissible to use antipyretics during the course of typhoid fever, no matter how high the temperature may be, if life is not threatened. One such case comes to my mind among a number of others. We have all seen cases which were particularly susceptible to drugs, to the application of external cold, to any kind of stimulant, and to opiates or alcohol in any form. The case which I have in mind is that of a young woman who was in the private ward of the Presbyterian Hospital, with typhoid fever running a regular course, but with excessively high temperature. It was impossible to administer any antipyretic. She was extraordinarily suscep-

tible to quinine, one grain producing serious nervous symptoms. The use of alcohol caused increased headache and violent nervous symptoms. The external application of cold and the external use of alcohol or other refrigerants produced shivering and great depression. Without my orders, on one occasion five grains of antipyrin were administered and such serious collapse ensued that it required the efforts of three of the internes for five hours to restore the patient to her natural temperature, and, in fact, to save her life. It was the most extraordinary degree of depression that I have seen from a single administration of any drug. The above is an example of a number of cases in which it is impossible to administer antipyretics. This is due to peculiarities of individual constitution. I think that physicians who are constantly calling attention to the use of antipyretics neglect to look for these peculiarities, which are present in cases frequently seen in private practice. I have now under observation an individual of highly nervous temperament, to whom I would not administer an antipyretic unless life was absolutely threatened.

It is in the latter stages of typhoid fever that I think antipyretics are especially dangerous, and must be administered with the greatest care. Two classes of such cases are worthy of attention. There is one class in which the temperature persistently keeps up to the full height, but there is abatement of all the other symptoms; lessening of the diarrhœa, cleaning of the tongue, diminution of the delirium, and even lowering of the pulse with increase of strength. We feel that the typhoid process is ended, but the fever nevertheless continues high. I will illustrate this with brief reports of two cases. In the first case, that of a lad ten years of age whom I attended during the course of an attack of typhoid fever, from the fifteenth to the eighteenth day of the disease the temperature ranged between 104° and 105.5°. The tongue had become clean, the stools had lessened in number and were gradually becoming formed, and I felt that the pathological process was at an end, and that if we waited a little the fever would probably fall. There was no dangerous symptom except the high fever. At

ten o'clock on the evening of the eighteenth day the temperature was 105°, and then began to fall until, at six o'clock in the morning, it was just below 96°, and required all the efforts of the father and the nurse to prevent what they thought to be a fatal collapse. The temperature remained below 96° for an hour, then began to rise, and by nine o'clock in the evening it was 104°. It remained at about this point for two hours, and then gradually fell until in the morning it was 96.5°. It then rose again, to be followed by another fall, but not to such a low point. There is no way in which this fall in temperature could have been anticipated. If we had applied external cold or administered an internal antipyretic I am quite sure that the collapse which occurred naturally would have been a fatal one. This sudden fall often occurs in typhoid fever in children, and it is one of the ways in which the fever subsides, the disease terminating by crisis.

In the second case—that of a young man aged seventeen years, who was treated in the private wards of the University Hospital a year ago, and who had a prolonged and severe attack of typhoid fever, the temperature keeping high—without warning or apparent cause a "nervous chill" occurred, followed by a great fall of temperature from 104.4° to 95.5°, the thermometer rising again to its former degree. The temperature kept thus for five or six days, and then gradually fell. I know of no way in which that fall of temperature could have been anticipated. I know that we were much tempted to use some active means to reduce the temperature, but, had this been done, it might have been serious for the patient.

I recently treated, in the Presbyterian Hospital, a man aged twenty-six years in whom the disease ran a perfectly normal course. He was treated with turpentine. The symptoms while grave were not alarming, and the temperature ranged between 103° and 104°. At one of my visits I found a falling temperature, which continued until the mercury registered 96.5°, being followed by a gradual rise with a subsequent decline by lysis. This is another case in which no one could have anticipated the fall of temperature, which, I am sure, was

a part of the disease, and not due to any cause which would produce collapse.

These three cases are examples that I have taken at random from an abundance of typhoid fever notes in which the administration of an antipyretic in the latter stages of the disease would have been dangerous.

Of course there is still another class of cases in which it would be dangerous to administer an antipyretic; that is, the class of cases to which Dr. Da Costa has particularly called attention, namely: cases in which the morbid process has terminated, but—on account of the exhaustion, or the long continuance in bed, or the limited diet—the temperature keeps up, or even begins to rise. Such a case was under my care last year. These have been spoken of as cases of bed-fever, and in these cases the administration of an antipyretic would not have the desired result. In these cases the fever is due to exhaustion, lack of food, and long continuance in bed. The use of stimulants, solid food, and getting the patient out of bed bring back the temperature to the normal.

The above are the classes of cases to which I wished particularly call attention in connection with the sometime danger of antipyretics in typhoid fever. I bring this essay to the College as much for criticism as for discussion, and, finally, in order to say that if physicians *will* give antipyretics in typhoid fever, let them be careful not to administer these drugs in the class of cases or under the conditions to which attention has been here called.

DISCUSSION.

Dr. H. C. Wood: I am inclined to think that the use of internal antipyretics is unnecessary and dangerous in typhoid fever. I do not believe, however, that the sudden collapse which is set down to the antipyretic is the direct or the immediate outcome of the action of these drugs. I am very sure that antipyrin itself is not a depressant to the heart. I have connected the needle of the cardiometer with a large artery of a dog with septic fever, and had it writing on the drum. Then I have given antipyrin in sufficient doses to reduce the temperature five or six degrees, and have found that the

writing on the drum was exactly as before. The fall of temperature had been accompanied by no appreciable depression of arterial pressure. I think that it is the fall of temperature which produces the collapse, due to the withdrawal of the stimulus of the heat. I cannot agree with Dr. Musser that it would be unsafe to have used external cold in the cases which he has mentioned. I believe that the cases would have recovered better if external cold and heat had been used properly. I believe that cases of typhoid disease die because the profession does not understand the proper use of external heat. If the case he mentioned, in which the temperature reached 105°, had had cold water applied earlier, I do not think that the temperature would have reached this point, and, therefore, the system would have been less exhausted, and the secondary fall would not have occurred. The proper use of external heat can always control these falls of temperature, but the method of employing hot-water bottles, hot bricks, and the like, is futile. I believe that by the use of the ordinary water-bed three-fourths full of hot water it would be possible to heat a corpse. A few days ago I had a case which illustrated this. A feeble gentleman, the subject of chronic enteritis, was at the end of the third week of typhoid fever. One morning I found that the temperature was 97° and falling. Hot water was scarce in the house, as the boiler was out of order, and in the forty minutes during which we were heating the water the temperature had fallen two degrees. The man had not been on the bed more than twenty minutes when, although he was sweating profusely, two degrees had been gained, and in thirty or forty minutes his temperature was normal. The bed should be only half-full of water, so that the patient will sink into it. The temperature of the water should be from 140° to 170°. The bed should be well covered with blankets so that the patient will not be burned. He is surrounded by this hot temperature, and such a mass of water will keep its temperature for twenty-four hours. When the temperature of the patient has risen sufficiently, you slide him on to the ordinary mattress, and, if necessary, he can be returned to the water-bed. In this way you can control the fall of temperature by hot water as readily as you can control the rise of temperature by cold water, and with less discomfort to the patient.

Dr. James Tyson: I believe that it has come to be generally conceded that the continuous use of antipyretics of the class represented by antipyrin in cases of continued fever is on the whole harmful on account of certain depressive influences due to such use of these drugs. I am inclined, however, to agree with Dr. Wood that it is not so much the direct effect of the remedy on the heart as the effect of the profuse perspiration induced by these remedies. I have had some interesting experience with the use of antipyrin in large doses continued for some length of time. I have given fifteen grains, three times a day, as recommended by the French school, for two weeks, with the view of controlling glycosuria, without any apparent serious effect upon the patient. I had to do, of course, with an afebrile affec-

tion, and we know that in such cases perspiration does not follow the use of antipyrin. In the few instances in which it has worked badly it has seemed to produce derangement of the stomach from the size of the dose. I rarely, however, now use these large doses, for the following reasons: In the first place, I was disappointed in its controlling effect; in the second place, where there was a controlling effect—as does occur in the milder cases—the same result can be accomplished by a small dose; and, in the third place, on account of the derangement of the digestion, which sometimes occurs. I have, however, come to the conclusion that the direct depressant effect of antipyrin is exaggerated, in the popular mind at least, if not in the medical mind.

Dr. H. A. Hare: I want to speak of one point which I think has been overlooked in the administration of the cold bath in typhoid fever, and which has only recently been brought before the profession in America with the force that it deserves; that is, the importance of applying friction or peripheral irritation while the patient is in the cold bath, in order to cause the heated blood in the centre of the body to come to the surface. As I have seen cold water applied, the patient is usually wrapped in a sheet and sprinkled with a sponge or an ordinary water bucket, or he has been put in a bath-tub and allowed to soak until the temperature falls to what is thought to be the proper degree. This is, perhaps, the common way. Under these circumstances we frequently find that the patient either goes into a condition of collapse or when removed from the bath the fever returns to the same degree as before. The hot blood which has been driven by the contraction of the capillaries to the central organs comes to the surface as soon as the peripheral bloodvessels dilate, the cold being taken away. By the application of friction, however, while the patient is in the cold water the tone of the peripheral capillaries is kept intact, and as I have said the hot blood from the centre of the body comes to the surface and is cooled and is returned. This prevents the internal congestions which have been reported in some cases. This method also possesses the advantage that the friends do not consider it so exposing to the patient that is rubbed while in the cold bath as when he is simply placed there and allowed to soak. With the use of frictions I have found that patients rarely object to the cold. Under the ordinary method the patient when put into the cold water often has what may be called a nervous chill, the cold causing an almost convulsive nervous spasm. If he is rubbed, and the sponging begun not too suddenly this chill is not observed, and the only objection that I have had made has been a complaint of the burning of the skin on account of rubbing too hard.

I have seen a number of cases, such as Dr. Musser speaks of, in which the patient apparently went into a collapse in the course of typhoid fever. This is frequently seen in children and I have also observed it in adults. At my last term of service at St. Agnes' Hospital a year ago, I had fifteen

cases of typhoid fever in which the temperature was subnormal from the beginning of the attack to the end, with the exception of the first few days It never went above 99°, and was generally 96° or 97°. In another class of cases, more like those mentioned by Dr. Musser, the disease ended by crisis. I now have a man in the Jefferson Hospital whose disease ended typically by crisis.

I agree with Dr. Wood and Dr. Tyson concerning the use of antipyretics in typhoid fever. Three or four years ago when I was studying these antipyretics with Dr. Wood and by myself, I was inclined to give them a great deal, but I found that the treatment of typhoid fever by antipyretics was like placing straw before a drowning man. If the patient is going to have a high temperature for three weeks it is of no use to lower it for half an hour and then have it return. Such treatment is simply giving the emunctories the trouble of eliminating drugs, deranges the digestion and possibly tends to produce cardiac failure. I have, however, never seen cardiac failure, after the use of antipyretics, which was not due to the lowering of the temperature. Dr. Brunton and others have shown that the lowering of temperature is like taking away whiskey from a chronic alcoholic, for heat is a most important stimulant to the heart and respiration.

Dr. Frederick P. Henry: The importance of friction during the use of the cold bath is insisted upon by Brand, whose experience of this method of treatment is probably greater than that of all observers here combined. For the purpose of making friction there is nothing better than the vegetable called the loofah. Some years ago I used the antipyretic drugs very extensively at the Episcopal Hospital, but soon abandoned them, and now I do not use them at all. In the experience of Brand there must have been many such cases as Dr. Musser has spoken of, where there was a tendency to sudden collapse and termination of the disease by crisis, such as occurs in pneumonia without any material danger, as a rule. Brand reports twelve hundred cases treated by cold baths, in his own experience and that of two or three others. Of this number twelve died, making a mortality of one per cent. These deaths occurred in cases which did not come under the cold-bath treatment during the first week, and Brand claims that if his rule had been observed, and the cases treated before the fifth day, there might have been no death. The opinion of Dr. Musser that if cold had been applied in his case the results might have been disastrous, is negatived by Dr. Brand's statistics, which must have included cases in which this tendency to sudden fall of temperature existed.

Dr. John K. Mitchell: There is one thing in regard to antipyretic drugs which has not been mentioned. It is an opinion, of course, not capable of direct proof, but my impression is that, where antipyretics are given constantly or as a treatment, the duration of the disease is decidedly lengthened. I believe that others have made the same observation. I recall that Von Jaksch, now of Prague, when I was in the Vienna Hospital with him, stated

his belief to be that the course was lengthened by persistent administration of antipyretics. My experience with the cold-bath treatment has been small, but entirely satisfactory. I have never seen collapse, and all my cases have been rubbed while in the bath. Twice I have seen extraordinary falls of temperature. In one case it fell from 105° to 92° without collapse or symptoms of heart-failure. The temperature was so low that I doubted the correctness of the observation, but it was tested with three thermometers which all registered alike. This patient had sixty-five or sixty-six baths during the five weeks' duration of his disease. On each occasion the temperature fell at least four degrees, and in this one instance thirteen degrees.

Dr. J. P. Crozer Griffith: Dr. Mitchell is undoubtedly correct in his opinion that the persistent use of the drugs of the antipyretic class may prolong the course of typhoid fever. Ehrlich, in 1887, called attention to the fact that the spleen remained enlarged an unusually long time in cases in which he had employed what he called "thallinization," and I think it was also he who first noticed that the whole course of the disease was lengthened by the administration of antipyretics continuously.

Dr. Musser's first case is of interest in that it has a very intimate relation to the question of the employment of antipyretic measures in general. I cannot imagine that a pathological process *per se* can ever be of advantage —that fever is a condition to be desired. While it was apparently true that his patient was in a better condition during the portion of the day in which fever existed, yet the state of depression which followed was, in my opinion, directly in proportion to the entire duration of the fever and to the elevation of the temperature. As usual in physiological processes, the over-stimulation was followed by exhaustion. If there had been no afternoon fever, there soon would have been no morning depression. It is for this reason that cold baths should be used as Brand recommends—that is, employed before the temperature reaches any great elevation. Brand advises, I believe, that the bath be given when the temperature reaches the neighborhood of 102° F. I can well see that in a case like Dr. Musser's, or in any case in the later stages of typhoid fever in which the temperature has reached about 105° F., the use of cold water may be followed by collapse.

The employment of the antipyretic drugs is, I think, still more liable to be attended by serious depression of temperature, and not only in typhoid fever, but in all other asthenic febrile conditions, must we exercise caution in administering them. It has been a matter of great interest to me during several years past to attempt to discover just how much good and how much harm could be expected from these drugs. I have been especially interested in the administration of antipyretics in phthisis. Now, it is perfectly possible—though it seems paradoxical—in a large degree to prevent sweating in this disease, and that by the very drugs which are usually so liable to produce sweating in febrile states. This is not accomplished, however, by giving the medicine when the temperature has already reached 104° or 105° F. If

we give it then, we only precipitate and intensify the fall of temperature which is apt, in any case, to occur in the later hours of the night; and we consequently produce greater sweating and exhaustion than if we had let the patient alone. I have tried the administration of small, divided doses of one of the antipyretics, commenced early in the day, a short time before I expected the sensation of chilliness to appear, and in many instances I have kept the temperature from rising to any extent, and, consequently, the sweating from following.

It is not the direct action of the antipyretics which does the harm, but it is the fall of temperature, whether this be the result of natural causes or of medication; and, if we prevent the rise, we shall, of course, have no fall.

I think that antipyretics should not be used simply to reduce temperature, but that some other indications should exist. I have seen patients—as we all have—preserve a high temperature through the course of a disease, and yet be apparently in excellent general condition. In other cases nervous symptoms become marked whenever the temperature is high. We are to be guided largely by the symptoms apart from the height of the temperature. I have through several years preserved careful records of the effect of antipyretics in febrile diseases in children, and have thus procured some interesting data, which I hope to present to the College at a future time. I am sure I have repeatedly removed threatening convulsive symptoms by the prompt administration of antipyretic drugs. Whether this effect was entirely dependent upon the reduction of temperature, or whether it was the result of some special action upon the nervous system, I am unable to say.

Dr. Musser: I quite agree with Dr. Wood and others in regard to the harm and danger of antipyretics, and in fact in regard to the uselessness of them, during the course of typhoid fever. I never used internal antipyretics in this disease, but I am quite in accord with him as regards the value of cold. While my experience with the method of Brand is too small to enable me to form any conclusion, yet the writing of the Germans and of Dr. Wood, Dr. Wilson, and others in this country is sufficient to convince me of its value. While cold is undoubtedly of value, I am also quite convinced that it is not necessary to use it in all cases. It is not justifiable in private practice unless we have the proper appliances, the proper means to apply it. I always have been opposed to the use of antipyretics in the management of typhoid fever, and the purpose of the few notes that I have made has been to call the attention of the large body of the profession to their dangers, for I am sure that it is quite common among the profession to use these drugs. Those of us who have the opportunity of observing cases in hospital practice are well satisfied that these drugs are dangerous. We have, however, all seen cases in which enormous doses of antipyretics have been administered, and there is no doubt that these drugs are often employed. It was with the intention of calling attention to the few condi-

tions in which the use of these drugs might be dangerous that I wrote this paper, in order to stem the tide toward antipyretics.

If Dr. Wood could tell us how to manage cold as conveniently as hot water it would be of great service to us. In private practice we often have great difficulty in securing proper appliances, and in obtaining the consent of the family to the use of this measure.

Dr. Wood: I have not given this question of the application of cold any consideration, but it occurs to me that possibly we could use the water-bed filled with ice-water, just as we use it filled with hot water.

OBSCURE FORMS OF GOUT.

By CHARLES W. DULLES, M.D.,
PHYSICIAN TO THE RUSH HOSPITAL, PHILADELPHIA.

[Read March 2, 1892.]

Some years ago a friend of mine, a physician, while travelling in Europe, was the subject of an obscure sense of ailing. He was not absolutely ill, he thought, but he was not quite well, and had a feeling of depression which he could not account for. Being in London, he made up his mind to consult Sir William Jenner. After a visit, at which Sir William subjected him to a careful physical examination, he was surprised at being told that his trouble was gout. His surprise finding some expression in his countenance, Sir William said to him: "You do not believe me, but you will find that I am right. You Americans know little about gout, because you do not see much of it, and what you see you do not recognize." Within a few days my friend awoke one morning with a most obvious attack of gout in one of his feet, and his respect for gout and for Jenner's diagnostic ability was considerably increased.

This story is so in the line of some recent observations of my own that I have chosen it to introduce a brief presentation of them and the reflections to which they have given rise in my mind.

Gout is a disease that, in the last few centuries, has been studied with great thoroughness by the English, the French, and the Germans; and it is probable that the earlier American physicians, following the teachings of older and contemporary English writers, and dealing with patients of habits very simi-

lar to those of their English ancestors, were alert to detect its presence. But I have the impression that Sir William Jenner did not exaggerate much when he expressed so strongly the idea that in our day gout is not taken into account by American physicians as it ought to be, while an indication as to the amount of attention it has received from the American authors of medical works may be found in the fact that the Index Catalogue of the Surgeon-General's Library at Washington contains nearly twelve columns of titles of books on gout, and among them I find only three books—and these not imposing—by American writers.

Within the past year three cases of gout—among others—have come under my care which strikingly illustrate the importance of recognizing forms of gout that depart from the picture usually regarded as typical. These cases have been so instructive to me that I think it may be of service to describe them.

The first of these cases is that of a gentleman of means, but actively engaged in a business that required much thought and involved serious responsibilities. He is about fifty-five years old, of full habit, and "a good liver," but free from all forms of intemperance. Near the end of last winter his son brought him to my office in a condition that is hard to describe; but one that each of us have probably often seen. He did not present any marked signs of disease, but he was pitiably unnerved and broken down. His appetite was poor, his sleep disturbed, his bowels somewhat irregular. He seemed so much to need mental rest that I suggested that he go away to Florida. I wished to examine his urine; but he slipped away to a health resort in North Carolina before I had an opportunity to do so. He came back in a few days feeling much better, and I did not hear from him for some time. Several months later, I was asked to see him by his regular family physician, and found that, after a short spell of feeling out of sorts, he had fallen one day on the staircase of his house in what was called a "faint." On inquiry, I found that in the "faint" the patient had continuously opened and closed one hand. In other words, his attack was not syncope, but a convulsion. His regular attendant and I agreed that we must study his urine, and on doing so I found in it a large number of crystals of uric acid. As he was at that time about to go with his family to Europe, I warned him in regard to the dangers of the gouty diathesis, and advised him how to avoid them as far as possible. A few weeks later he wrote me from Paris: "You are a good prophet; I have just had a pronounced attack of gout in the foot."

Since then he has returned to his home and had several more attacks of equally typical character.

The second case was that of a lady living in the quiet of an admirable family. Nearly two years ago she had an attack of "grip" when the epidemic was most severe in this country. After that she was never quite as well as she had been before, and about six months ago she began to suffer with pains of a neuralgic character, which were especially severe in the thighs and in the lower part of the back, and which appeared also in the chest-wall and elsewhere. Some of these closely resembled sciatica. She was not under my care; but once or twice I was called to see her in the absence of her regular physician, and once when I gave her a subcutaneous injection of one-quarter of a grain of morphine, I was much impressed with the fact that this injection of morphine quieted her and kept her asleep or sleepy for fully thirty-six hours. Some time after this, while away from home, she suddenly developed symptoms of advanced disease of the kidneys; she was brought home and died in twenty-four hours, with waxy complexion, œdema of the face and legs, oppressed breathing, and a general appearance of the phenomena of contracted kidneys. The patient's knuckles were knobbed, and I have no doubt, from a long acquaintance with her, from observations made on occasional visits, and from the manner of her death—which I watched for ten hours almost without intermission—that she died of contracted kidney due to gout.

A third case of gout which did not present the most conspicuous manifestations of this disorder, was that of a physician about forty-five years old, a man of means and not in practice. Last June he sent for me, and when I came, he said: "I am afraid to see you." I said: "Why?" He replied: "I think I have an aneurism of the aorta; and I fear you will confirm my opinion." He then described symptoms, some of which are found in cases of aneurism of the aorta, and submitted to a thorough physical examination. I found no evidence of aneurism whatever, and told him so; when he naturally asked me how I accounted for his symptoms. In preparing an answer to this query, I inquired pretty fully into his history and examined his urine. The former gave distinct points consistent with a theory of gout, and the urine showed an abundance of crystals of uric acid and oxalate of lime, some kidney epithelium, and no albumin or sugar.

I then told my friend that I believed his symptoms were due to gout, and advised a regimen in accordance with that theory, and the use of a natural mineral water that I have found advantageous in similar cases. In a few days he felt better; in a few weeks he felt well; and to-day he is apparently in perfect health. A year ago he was unable to enjoy anything; to-day he is full of spirit and enjoys everything. A year ago he could not walk a hundred yards on a slight ascent without dyspnœa and precordial distress; now he walks miles with ease and pleasure.

It would be a work of supererogation for me to dwell upon the symptoms and course of frank gout, for these are familiar to every practitioner of experience, especially in our older cities. In throwing off the British yoke the American colonists secured no independence from the diseases of their British progenitors; and the tendencies that our forefathers inherited, though they may have been kept in abeyance while the nation was passing through the arduous and heroic epochs of its early history, seem to be finding more pronounced expression, now that the number of persons living in comparative ease or actual luxury is increasing. As it was in Greece and Rome, so it has been in England and on the continent of Europe, and so it is likely to be in America. Abernethy's advice to "live on a shilling a day and earn it," indicates a road to health which is constantly being deserted by Americans; and as they do so, the diseases of over-feeding and inadequate muscular exercise must become more conspicuous among us. For this reason gout is likely to secure more and more victims in this country, and consequently it is well that American practitioners should be prepared to recognize not only its striking forms, but also those which are irregular or obscure.

In his classical work on *Gout and Rheumatic Gout*, Garrod says that, while gout in its most marked and typical manifestations is exceedingly prevalent in England, "in its lurking and undeveloped forms it is probably still more so, and exercises a considerable influence over the character and progress of other disorders." From this, it is a natural step for him to emphasize the importance of properly distinguishing gout from other diseases, of recognizing it when it is present, and at the same time avoiding the error of attributing to gout what are really manifestations of other disorders. Everyone who becomes especially interested in a particular disease is in some danger of falling into this error, and the more likely to do so as the idea of anomalous forms of that disease takes possession of his mind.

In determining the diagnosis of gout we must study the history of the case—the hereditary tendency of the patient, the

existence of predisposing or exciting causes, and the symptoms. In retrocedent gout—that is, the development of visceral disorders or lesions upon the disappearance of gout in the joints—the case is simple, although Garrod aptly quotes a remark of Watson that "so-called *gout* in the stomach has sometimes turned out to be *pork* in the stomach." There is undoubtedly a form of dyspepsia in persons predisposed by heredity to gout that may justly be attributed to this disease; and this form of dyspepsia is sometimes relieved upon the appearance of gout in a joint, while treatment suitable to gout proves curative of it.

Gout of the heart is often associated with gouty dyspepsia; but Garrod speaks of cases in which palpitation, and irregularity of rhythm have been produced by the state of the blood. Dyce Duckworth, in his elaborate work on *Gout*, emphasizes the importance of what may be called minor symptoms of disturbed action of the heart, such as giddiness and dimness of vision, in gout. A case of my own has impressed me with a similar conviction. I myself am inclined to believe that certain forms of angina pectoris are dependent upon the gouty dyscrasia. I have seen three cases in which long-continued disturbance of the functional action of the heart seemed to me to be altogether dependent upon gout, to which the patients were liable by heredity or by their habits, and of which they presented other manifestations. This is a subject which I think deserves very careful investigation, so that we may ascertain just how far we may look to the gouty habit to account for heart symptoms which are not otherwise easy to explain.

The effect of gout in producing cough, dyspnœa, and even asthma, is recognized by almost all authors; but there is danger, I think, of attributing such disorders unjustly to gout, with which they may be mere coincidences. At the same time it is proper to recognize that, if not directly induced by gout, they may be exaggerated or made less amenable to ordinary methods of treatment by reason of the gouty dyscrasia. Examples of this have certainly occurred in my practice.

The kidneys are probably the organs which are most likely

to undergo serious alterations in consequence of unsuspected gout, and a recognition of this fact is of the utmost importance. Draper, in his admirable article on gout in Pepper's *System of Medicine*, speaks forcibly of the insidious character of gouty nephritis, and states that gout of the kidney is the most common cause of death in this disease. The observations of other writers on gout are in accord with this statement, and it can hardly be dwelt upon too strongly in studying the possibilities of gout. In one of the cases briefly described above I believe that a careful study of the history of the patient, and a due consideration of the possibilities of gout, would have prevented a very erroneous prognosis on the part of one physician who treated her, and perhaps have led to a more appropriate line of treatment.

In this particular case a consultant of large experience was quoted to me as treating with apparent unconcern the state of the kidneys, because the quantity of albumin found in the urine was small!

Such a statement suggests either an error in the account of what was said, or a serious misapprehension of the significance of the presence of small quantities of albumin in the urine. On the other hand, it is possible that large quantities of albumin may be found in the urine during comparatively slight attacks of gout. Very recently I had under my care a young lady who was suffering with an attack of podagra, very well defined, but of moderate severity, whose urine contained enough albumin to make, with underlying nitric acid, a coagulum one-third of an inch thick. This patient presented no other symptom of kidney disease—her urine was repeatedly and thoroughly examined—and she made a good recovery from her attack of gout.

Temporary glycosuria is another evidence of faulty nutrition or disordered excretion to be found in some cases of gout. Dyce Duckworth points out the danger of mistaking this phenomenon for diabetes mellitus, and especially of communicating this erroneous belief to the patient or his friends.

The condition of the system called lithæmia is probably, in

many instances, only a form of gout, the typical symptoms of which it may present. Da Costa, in his work on *Medical Diagnosis*, speaks of the close connection of gout with lithæmia, and says: "Indeed, the excessive formation of lithates and the dyspeptic symptoms, with the heartburn and eructation, the signs of functional derangement of the liver, the vertigo, the mental gloom or the listlessness and indisposition to exertion, the cramps in the legs and muscular twitchings, the neuralgic attacks, the restless nights, the palpitations of the heart and its irregular beat, are in many but the precursors, although, it may be, the long precursors, of a regular outbreak of gout."

The influence of gout upon the skin has been especially emphasized by certain French observers. Draper cites Bazin (*Affections génériques de la Peau*, Paris, 1862) as describing functional derangements of the skin dependent upon gout, such as excessive perspiration, seborrhœa, alopecia, pruritus, urticaria, and erythema. Eczema is a well-recognized manifestation of the gouty diathesis, and Draper calls attention to the frequency of acne in persons predisposed by heredity to gout. Garrod, in 1881, stated that he regarded eczema as the special skin lesion of gouty subjects. I have noticed a peculiar fetid, or rather a sour perspiration in a few persons belonging to gouty families, some of whom in time developed frank attacks of gout. As connected with the skin, it is interesting to note that Dyce Duckworth speaks of spontaneous loosening and falling out of the teeth as a manifestation of gout.

One of the most important manifestations of obscure gout is neuralgia. There is hardly a writer on gout who fails to call attention to this feature of the disease, and yet I believe it is by no means properly appreciated. The neuralgia of gout may affect any region which an ordinary neuralgia may invade, and may easily be misinterpreted. This is especially true of the sciatic region, so that what is only a manifestation of gout may be called sciatica. One feature of such cases which may be noted, is the fact that the neuralgia of gout appears in various places at the same or at different times. The ham, the calves of the legs, the loin, the side of the chest, the arm, and

the region of the fifth pair of nerves, may be the seat of severe and even agonizing pain; and this very shifting of location ought to warn the physician to carefully consider the possibility that gout is the cause of the phenomena.

Other manifestations of gout in the nervous system are vertigo, loss of consciousness, and even epileptiform convulsions. These are rarely of a very pronounced character, but Garrod mentions one case in his observation, and one of the cases I have described presented this symptom. The relation of gout to this and other forms of cerebral disturbance is not yet clearly defined, and it would probably be useful for men of large experience to study their cases with especial reference to this relation. In my practice I have observed that gout has on some subjects a peculiarly depressing influence, so that they are easily disheartened, and lack vigor of will. Laycock has called attention to its influence in producing hysteria, and many authors connect it with hypochondriasis. Dyce Duckworth has been so impressed with the nervous features of gout that he defends the hypothesis that gout is a neurosis. The same author speaks of the occurrence in gout of violent pains in the muscles—as, for example, cramps in the legs.

These remarks do not by any means cover all the manifestations of gout which may be misunderstood, but which, if duly considered, may lead to its diagnosis when what are universally recognized as its typical symptoms are absent; but it is hoped that they may serve to attract attention to, and, perhaps, also throw a little light on a subject which is of some consequence in our study and treatment of disease.

As already stated, I have the impression that in this country gout often escapes diagnosis, and that it would be better for our patients if we were on the alert to detect its less familiar features. We do not want to deceive ourselves by a too ready disposition to find gout where it is not actually present, but to keep in mind that gout may be the cause of a great variety of symptoms, and to know what some of these symptoms are may at times make our diagnosis much more satisfactory and our treatment much more successful than they would otherwise be.

DISCUSSION.

Dr. J. H. Musser: I have listened with pleasure to the interesting paper of Dr. Dulles, and arise only for a moment to take exception to the idea which I fear is cast forth that gout in its obscure forms is not recognized in this country. I am quite sure that we, as individuals, are on the alert for obscure forms of gout, although we may not call it by that name. We are constantly looking for lithæmia, or lithiasis, or the uric acid diathesis. We are constantly arranging the diet and hygienic details of individuals with this idea in view. Ophthalmologists constantly see cases of gouty disease of the eye, and those who work in nervous diseases constantly have in mind this fundamental condition. It is only to take exception to this one point that I rose to speak.

Dr. James Hendrie Lloyd: I have recently made an interesting observation on a gentleman who has been under my care for a number of years. He had a characteristic attack of gout in the large toe several years ago. It is hereditary. He recently came to me complaining of severe headache, which was always worse in the morning. I subjected him to a very careful examination, and sent him to Dr. Oliver, who examined the eyes with negative results. I was beginning to fall back upon the idea that the headache was due to gout, and to direct the treatment with that idea in view. About that time the patient wore a close-fitting shoe one day with a fold of the stocking making compression on the proximal end of the great toe. This started a furious attack of gout which laid him up in the house. It confirmed the diagnosis. The headache improved at once, and he has been quite well since.

Dr. D. D. Stewart: I would ask Dr. Dulles if he makes use of Pfeiffer's test for latent gout. This is: Take a specimen of the twenty-four hours' urine and pass it through a filter, subsequently dividing it into two equal parts. One part is passed through another filter in which chemically pure uric acid is placed. The filtrate is then examined for uric acid. If the patient is gouty it is said that there is scarcely any uric acid found, whereas in the other specimen, which has not been passed through the uric acid filter, there is a normal or an excessive amount of uric acid. In those who are not gouty the uric acid passed through the uric acid filter is not taken up and the same amount of uric acid is found in both specimens. Sir William Roberts recently reported on this test, but did not find it reliable. He preferred to bottle specimens of the twenty-four hours' urine and put in a warm room. If it is from a gouty patient there is a deposit of uric acid in a few hours.

I should like to suggest that, in place of colchicum, which has a tendency rather to cause retention of uric acid in the blood, piperazine should be em-

ployed. This has a wonderful solvent action on uric acid. I recently prescribed it in the case of a young girl who undoubtedly has a stone in the kidney. Her cousin was operated on by Dr. Keen last year for the same trouble. This girl has typical symptoms of uric acid calculus. I gave her the various vegetable salts of potassium without affecting the urine, which had diminished to three-fourths of a pint in twenty-four hours. Piperazine is now being given; after taking it for ten days she stated when she last saw me that she had passed five pints of urine in less than twenty-four hours, and that all the symptoms had diminished, and the pain was much lessened. This remedy will doubtless prove most valuable in obscure gout or gout of any sort through its wonderful solvent effect upon uric acid. The urate of piperazine is soluble in fifty parts of water, while the urate of lithia requires three hundred and sixty-eight parts. The only objection to the drug is its expense. The cost is about nine dollars an ounce, but as the dose is only two to five grains three times a day, this is not in reality so great.

Dr. Dulles: I am sorry if what I have said has seemed to reflect upon the acumen of medical practitioners in America. I certainly did not mean that it should; but I feel that the subject of obscure forms of gout has not received such full or formal attention in this country as to make it likely to be generally and fully understood. For this reason I have felt that it would be worth while to make it the subject of a paper, and to call the attention of the Fellows of the College to the matter.

FORMS OF PSEUDO-TABES DUE TO LEAD, ALCOHOL, DIPHTHERIA, Etc.

By JAMES HENDRIE LLOYD, M.D.,

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[Read March 2, 1892.]

Locomotor ataxia is one of the best-known diseases. Its classic array of symptoms, so familiar to all, occur, as a rule, with a precision of type and regularity of sequence which admit of but few errors. Its history and course have been written so frequently and so well that little remains to be said; while, unfortunately, its prognosis admits of but such a frank statement of despair, that it has long been described as one of the most inveterate and progressive of diseases. Locomotor ataxia, however, like many other diseases, has its counterfeits. All is not necessarily lost, as was once taught, to all men who stagger in the dark, whose knee-jerks are abolished, and who have pain and numbness in the legs. These counterfeits, doubtless, are in the minority to the true ataxics, but that they are more prevalent than was formerly supposed is at least probable; and that it is highly important for the welfare of the patient and the credit of the physician to differentiate them, is apparent. This paper will discuss especially the etiology and diagnosis of these pseudo-tabes, and will be based largely upon personal observation.

In 1884 Dejerine[1] described a disease which he called *Nervo-tabes Peripherique*, and gave the clinical histories and autopsies

[1] Arch. de Physiologie Norm. et Patholog. 3e. sér., iii. 1884.

of two patients whose disease he had diagnosticated in life as locomotor ataxia. Both of these patients had incoördination, anæsthesia, abolished knee-jerks, slight atrophy, and paresis, without eye or bladder symptoms. They both had used alcohol to excess. The autopsies revealed very pronounced inflammation of the cutaneous nerve-endings, slighter changes in the intra-muscular nerves, and no change whatever in the spinal cord, nerve-roots or ganglia. Dejerine had not found any analogous cases reported at that time in medical literature, and deserves credit for demonstrating for the first time the striking resemblance of these obscure cases to locomotor ataxia. About the same time, however, Dreschfeld[1] described a type of alcoholic ataxia characterized by incoördination in the gait, absent knee-jerks, and lancinating pains, but without atrophy or paralysis. His clinical picture was strikingly like that of Dejerine, but was not supplemented by the same careful post-mortem findings. That same year Krüche wrote a paper on "Pseudo-tabes in Alcoholics."[2] But earlier than all of these, Samuel Wilks, in his lectures in 1878, described the drunkard's paraplegia, and said that some of these cases resemble closely locomotor ataxia. Wilks believed that the lesion was in the spinal cord, but he wrote before multiple neuritis was much more than dreamed of.

Still earlier (1867) Leudet[3] wrote an elaborate paper on chronic alcoholism, in which he described the same combination of symptoms—anæsthesia, hyperæsthesia, disordered gait, and paresis—and in fact came near to describing what we recognize to-day as polyneuritis. In very recent years the literature of multiple neuritis has grown to a vast extent. It has become, therefore, more and more necessary to differentiate the various types of the disease and to recognize its many causes. With this motive, Leyden in 1888 wrote a monograph in which he distinguished five forms, one of which he named

[1] Brain, July, 1884, p. 201.

[2] Deut. med. Zeit., 1884, No. 72, p. 229.

[3] "Etude clinique de la forme hyperesthésique de l'Alcoolism chronique, etc.," Arch. gén. de Méd., 1867, vol. i. pp. 5–39.

the sensory form, under which heading he grouped the acute ataxics, and borrowed for them, from the earlier paper of Dejerine, the title of *neuro-tabes peripherica.*

It may be said in general terms that all these cases of pseudo-tabes or acute ataxia are instances of a multiple neuritis of this sensory form of Leyden, and that most of them probably have a morbid anatomy similar to or identical with that demonstrated by Dejerine, *i. e.*, an involvement especially of the periphery of the cutaneous nerves. Hence the most common symptoms in these cases are disorders of sensation, especially anæsthesia and paræsthesia, with ataxia and abolition of the knee-jerk. To these must be added symptoms, in varying intensity, of involvement of the motor nerves, never perhaps quite absent, but often requiring a careful and expert examination to detect. From these ataxic cases all degrees of severity occur up to the well-recognized cases of multiple neuritis with general involvement of the sensory and motor nerves. The eye and bladder are seldom involved in these ataxic cases, but important exceptions occur. Of the great viscera, the heart and kidneys are most apt to suffer.

In some cases it is quite impossible to at once arrive at a correct diagnosis. In doubtful cases the history alone may determine the diagnosis; and this history, with certain attendant phenomena—as, for instance, the blue line on the gums in lead cases, tachycardia in alcoholic cases, and paralysis of accommodation in post-diphtheritic cases—should always be most carefully investigated. By far the most frequent causes in my observation are alcohol, lead, and diphtheria. Others report arsenic, and such infectious processes as variola, typhoid fever, tuberculosis and syphilis; also wasting diseases, as diarrhœa and dysentery. Bartholow once reported cases of paralysis following bowel disorder, which were probably examples of neuritis. Syphilis is now believed by many to be capable of causing a neuritis; indeed, it may be a question whether some cases of so-called syphilitic locomotor ataxia, cured by mercury and iodides, have not been cases of peripheral neuritis. Dejerine, in another paper, described the onset of a fatal multiple

neuritis in a morphine-taker; and I have myself seen amblyopia accompanied by anæsthesia of the feet and legs in an excessive smoker.

The eye-symptoms are the most reliable for purposes of differentiation. None of the forms of multiple neuritis, except the post-diphtheritic kind, have, as a rule, any affection of the internal or external ocular muscles. I have always looked for them in vain in alcoholic and lead cases. In diphtheritic paralysis, however, the ciliary muscle is often paralyzed, while the light-reflex remains—the very opposite to the Argyle-Robertson pupil seen in locomotor ataxia. At the same time there is apt to be paralysis of some of the external ocular muscles, causing strabismus—almost always associated with paralysis of the velum palati. In lead poisoning optic neuritis is sometimes observed.

The functions of the bladder and sexual apparatus are not often affected in any form of polyneuritis, especially the ataxic form. The only case in which I have seen impairment of the bladder was in a woman with grave alcoholic paralysis, with mental symptoms and weak, rapid heart. The involuntary passage of urine in her case was the result of her mental condition. In chronic inebriates the sexual appetite rather than the sexual power is impaired. On the other hand, locomotor ataxia, as is well known, is likely to exhibit, early in the case, impairment of sexual power and of the expulsive power of the bladder.

It is most probable that ataxia or incoördination, whether in true tabes dorsalis or in pseudo-tabes, is caused by the impairment of sensation. However caused, it is very similar in the two conditions. We have at present in the Philadelphia Hospital a man suffering with a severe grade of multiple neuritis, with wasting of the muscles of the extremities. He has an alcoholic history, although, strange to say, his disease developed rapidly after a sunstroke. This man has diminished and sluggish, not abolished, knee-jerks. His gait is typically ataxic, with elevation and flapping of the feet. He cannot stand an instant with his eyes closed; in fact, he has such a

condition of *astasia* that sometimes he is compelled to keep walking to avoid falling. His eyes are normal. It has been claimed by some that the element of muscular weakness, in cases of neuritis, admits of a distinction being made between the ataxia of the two conditions, as in multiple neuritis the ataxic movement is evidently more feeble than in tabes dorsalis, and the foot is lifted higher because of the paralysis of the extensors. But in the cases of pseudo-tabes, muscular weakness is often not conspicuous, and hence this distinction is not possible. I have recently put side by side a case of acute lead-poisoning and a typical case of locomotor ataxia for the purpose of comparing the ataxic movements in the arms, which are very marked in both cases.

The cases are as follows :

Male, aged thirty-eight years. After four months' exposure in a lead factory he began to have colic, constipation, vertigo, and headache. He then had several convulsions, and passed into a state of lead encephalopathy, from which he is recovering. He has no paralysis of the extensors, but the shoulder muscles and biceps are paretic. He has some patches of anæsthesia on the arms, hands, and legs. His walk is ataxic, and the movements of his arms markedly so. He has the blue line on the gums, and the margins of the optic discs are slightly clouded. He has no albumin or casts in the urine.

This man's case has been a grave one, and admits of no confusion. The ataxic movements of his arms, however, are almost identical with those of the second case, as follows :

Male, aged thirty-eight years. He may be called a case of rapid acute locomotor ataxia. It began with numbness and pricking sensations. The patient was soon suffering with fulgurant pains; he then had ataxia, with inability to walk in the dark. The arms are involved as well as the legs. The knee-jerks are abolished. For a year he has had nocturnal incontinence of urine, and when the bowels are overloaded some paresis of the sphincter ani. The pupils are unequal and do not respond to light. There are irregular discs, with contracted arteries. There is apparently some latent iritis, and there is a posterior synechia. The man has had syphilis.

The ataxic movements in these two men are so similar as to attract attention, the only difference being that the man with lead poisoning takes longer to perform a given action than the

tabetic, probably because of the element of muscular atrophy in his case.

Motor symptoms, other than ataxia, are not conspicuous in cases simulating tabes; in fact it is the absence of marked paralysis in these cases of multiple neuritis that more than anything causes them to resemble cases of true locomotor ataxia. This was so in Dejerine's cases already referred to, in which the autopsies showed the principal changes to be in the cutaneous nerves. These are the cases which Leyden classes in his "sensory" forms, and calls acute ataxics. They are more often alcoholic and diphtheritic cases than any others. It is most probable, however, that none of these cases is entirely exempt from paresis in some muscles. Careful examination usually reveals some loss in muscular masses, and electrical tests sometimes solve a doubt. Paralysis may be slight, especially in the large muscles of the limbs, and may not be conspicuous when the patient is lying down, but when standing up or when executing fine movements with the fingers and toes the impairment is more marked.

I recall the case of a man in middle life who had diphtheritic ataxia, with anæsthesia, but who had so little paralysis that he was able to be about without attracting attention. Such cases in middle life are especially apt to be diagnosticated as cases of locomotor ataxia.

In diphtheritic cases acute sensory symptoms, such as pain on pressure over nerve-trunks and on handling the limbs, are not nearly as marked as in alcoholic cases. In all cases, however, presenting true ataxia from whatever cause, I believe a diminished or altered sensory function can be demonstrated by careful examination.

Rapidity of evolution is not an absolutely sure sign that the disease is a multiple neuritis, because true tabes may evolve rapidly. I have under observation a case of apparently true tabes in a ship-carpenter which began and continued as follows:

Two years since, while crossing the equator, being much overheated, he took a shower-bath, by having buckets of water thrown over him. He was in good health at the time. Three hours later he began to have tingling sen-

sations in the extremities, which he says were at first confined to one side. In a few days he was paralyzed. Later, the other side was affected, but never so badly as the right side. At present he has a very ataxic gait, pains in the legs and back, and abolished knee-jerks. One pupil is twice as large as the other, the dilated iris being immobile, and the other showing the Argyle-Robertson phenomena. He cannot stand with his eyes shut. His control of his bladder and rectum is impaired, and one ankle is enlarged somewhat like a tabetic joint. The muscular nutrition in the arms and legs is not bad, and his grip in each hand registers 80°. The man has had destructive nose disease, but denies syphilis.

This patient's history suggests neuritis, but his present state is so exactly that of locomotor ataxia, with eye and bladder symptoms, that the conclusion is almost forced that such is the disease.

The case of the patient from whose cord and nerves I present sections, has interest because the diagnosis of locomotor ataxia was actually made by an expert clinician.

I. L., aged fifty-eight years, painter by trade. He had been exposed to lead for thirty years. He had a very clear history of many attacks of colic, constant constipation, and several attacks of wrist-drop. He had also the blue line on the gums, and that frequent result of chronic plumbism, a contracted kidney, as shown by casts, albumin, and the specific gravity of his urine. No lead could be detected in his urine. He had been an excessive drinker, but had never had syphilis. The diagnosis with which he was sent to me had been made largely upon the ataxic gait, swaying with closed eyes, abolished patellar reflexes and numbness, tingling and retarded sensation in the soles. I found his real condition as follows: In addition to the above very evident symptoms, he had well-marked paresis of both the extensors and flexors of the forearms and hands, as well as the biceps and deltoid muscles. On voluntary motion there was a tremor rather than ataxia of the arms and hands. There were paræsthesia and areas of anæsthesia on the hands, legs, and soles of the feet. The legs and arms were slightly contractured. The external and internal eye muscles were normal. Sexual power was normal, and the control of the bladder was perfect. The gait was ataxic, but flapping of the feet was not marked. The hands and feet were slightly œdematous. There was partial reaction of degeneration and abolished faradic contractility in all the paretic muscles. Optic atrophy was noted by Dr. Gould—no doubt the result of optic neuritis due to lead. The diagnosis was chronic lead-poisoning, with multiple neuritis and contracted kidneys. The patient died in a few weeks in uræmic coma. The sections show quite extensive inflammation, or degeneration due to inflammation, in the several nerve-trunks examined,

with involvement, rather diffused, of some areas of the cord, notably in the lumbar region. The systemic changes of locomotor ataxia are not present.

In studying this case, in which it seems probable that the cord was involved later than the nerves, I have been reminded of the suggestion made by Leyden, that true tabes dorsalis may be secondary sometimes to a peripheral neuritis of the sensory or cutaneous form. This is still further demonstrated in a recent study by Pal,[1] who, in a series of cases of multiple neuritis due to alcohol, arsenic, typhoid infection, etc., found changes, apparently secondary, in the posterior columns and other parts of the cord. It is also well to recall that locomotor ataxia is complicated sometimes with an acute onset of multiple neuritis.

DISCUSSION.

Dr. Charles K. Mills: I have, of course, seen a number of cases of pseudo-tabes, but I do not feel that I have much to add to what Dr. Lloyd has said; but it may be worth while to discuss one or two of the points which he has presented, and also to call attention to the diagnostic features which separate pseudo-tabes and the ataxia of posterior sclerosis. Dr. Lloyd has spoken of, and perhaps laid sufficient stress upon, the fact that multiple neuritis and disease of the cord, and, indeed, of the entire cord, frequently concur. In these cases there is, of course, increased difficulty in diagnosis; but one difference holds as between cases of this kind and those of the ordinary form of cord disease—namely, that they are inflammatory, usually of acute character, and more curable. My own belief is, that when we have lancinating pains, as we occasionally have in pseudo-tabes, we almost invariably have associated disease of the cord. In a recent paper in the *International Magazine*, I record the history of a case which I studied for several years, and which I had the good fortune to see when the patient was first admitted to the hospital. This patient was then undoubtedly suffering from an acute and severe type of multiple neuritis. The symptoms were typical. After a time these symptoms disappeared under rest and treatment, but he began to present the symptoms of true posterior sclerosis, which remained until his death, several years later. He died a typical case of the ordinary type of posterior sclerosis. My own experience is, that there may be eye and bladder symptoms in true

[1] Wien. klin. Wochenschr., 1891, abstracted in Journal of Nervous and Mental Diseases, March, 1892, p. 225.

neuritis, but this is comparatively rare. I believe that a difference exists between the gait of the posterior sclerosis case and that of the multiple neuritis case in which the cord is not involved. In the paper to which I have just referred, I have a picture of the patient. In multiple neuritis in the chronic stage we have a combination of symptoms of paralysis with irregular anæsthesia due to the neuritis, and not to the cord disease. In posterior sclerosis we have ataxia with retardation of conduction of sensation, and at times perhaps true cutaneous anæsthesia. Anæsthesia with motor loss will give a gait and other symptoms in the arms which are similar to, but not exactly the same as we see in posterior sclerosis. It is difficult to describe the difference, but it is a difference which depends upon irregular paralysis with some anæsthesia. If we study these cases closely, we will find, for instance, that the patients are not able so well to make use of the limbs forcibly in a sitting or lying position. We will be able to separate different degrees of paralysis in different groups of muscles. In the ordinary case of ataxia, unless in advanced stage, this is not present. There is a peculiar flapping of the feet in these cases, which is not the throwing about of the feet in the ataxic case. This is a matter of interest, and we may after a time learn at sight how to tell these cases apart.

I have been called in several cases where the diagnosis of true tabes has been made by a general practitioner of experience, and where the cases have turned out to be curable, and some are now well. Dr. Lloyd has called attention to the diagnostic symptoms, but did not summarize them. Among the most important are the absence, although not invariably, of bladder and of eye symptoms; the combination of paralytic loss with anæsthesia, and the presence in the cases of neuritis of reactions of degeneration which are not present in ordinary true sclerosis. Another point of considerable value in the diagnosis is the frequent presence of mental symptoms of a peculiar type at certain stages, particularly of the alcoholic pseudo-tabes. I saw only to-day, in my office, a patient of this kind, a comparatively young man who had a history of excessive use of alcohol. He had lost knee-jerks, slight ataxia, some anæsthesia, and a very decided mental change and deterioration. The history of the case and the absence of lancinating pain enables us to approach a diagnosis of pseudo-tabes. The existence of dermal neuritis is a matter of interest. I believe that dermal neuritis may exist when the trunks of the nerves are not affected, or practically not affected. I have seen pseudo-tabes in several other affections besides alcoholism, plumbism, and diphtheria—notably, in a case or two of diabetes; and also in syphilis I have seen a few cases which closely simulated true ataxia.

Dr. J. P. Crozer Griffith: I am glad that Dr. Mills referred to the differences in gait in the way that he did, as I was about to ask a question in regard to this matter of those who constantly see much of these forms of

nervous diseases. I remember that five years ago, in a very interesting article in one of the journals, Ross called attention to the likeness in many cases between the symptoms of multiple neuritis and those of locomotor ataxia, but also to the fact that there were differences in the gait in the two affections. In the case of neuritis there existed a certain dropping of the toe in walking which perhaps could not be seen unless the observer took his position behind the patient. He stated, also, that the patients were unable, while sitting in a chair, to raise the toe from the floor while retaining the heel upon it.

Dr. Lloyd: In reply to Dr. Griffith, I would say that I refer in my paper, although briefly, to the fact that fine movements of the feet, toes, and hands are likely to be affected in cases of pseudo-tabes. Ross calls attention to the fact that such patients could not lift the toe with the heel on the ground. This is only one example of this interference with the fine movements. In nearly all cases of multiple neuritis the extensors are more paralyzed than the flexors. The sensory symptoms and the ataxia are more apt to attract the attention of the general clinician, and he does not study the finer muscular movements sufficiently.

In reference to syphilis, I refer to one or two cases which I have read or heard of where the claim has been made of the cure of syphilitic tabes. When we have true locomotor ataxia with eye symptoms, whether syphilitic or not, I doubt whether there is any chance of recovery. I think that in some cases of so-called syphilitic tabes in which cure has been claimed the disease has not been a true tabes, but a pseudo-tabes due to degeneration of the peripheral extremities of the nerves. It is well to recall that Erb has recently proved by the study of a large number of cases that the great majority of cases of true locomotor ataxia have a syphilitic history. The fact that a man with true tabes dorsalis has had syphilis does not justify his physician in entertaining or encouraging the hope of a cure.

TREATMENT OF SACCULATED AORTIC ANEURISM BY ELECTROLYSIS THROUGH INTRODUCED WIRE.

Report of a Case.

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[Read March 2, 1892.]

The therapy commonly adopted for the cure of aortic aneurism, whether of the arch or the descending portion, usually offers so little promise of success, that any method suggested to supplement or substitute this, if attended with little risk to the patient and supported by the slightest clinical evidence, merits careful consideration. The frequent utter inutility of medical treatment in cases of aneurism which, from their situation or character, are insusceptible of extinction by deligation or by compression, necessitates an earnest search for another method.

As the cure of aneurism can only result through obliteration of the sac cavity by the formation therein of firm thrombi, which in process of time undergo organization with contraction of the sac wall, so all plans of treatment suggested have as their basis the production of organizable clot. The ideal method of promoting this would be that of introducing into the sac a harmless substance that would act by virtue of its vital or chemical properties; such a substance has not yet been found among the medicinal hæmostatics. Experiments

made by the late Dr. Wooldridge[1] tend to indicate that it may exist in lecithin or in tissue fibrinogen, that the fibrin factors of the blood may, under skilful manipulation, be so modified that these can be introduced without risk of the coagulum induced spreading beyond the sac boundaries into the vessel. As yet, this hope has not been realized.

Multiple galvano-puncture, with the use of strong currents, the anode the active pole, the cathode externally; in brief, the process conducted in a manner conducive to the best results with least risk, if good results were possible, has, beyond doubt, proved a failure in the therapy of aneurism.[2] Although the method is practically devoid of danger when carefully carried out, it is also, it must be added, apparently practically devoid of benefit; for though firm coagula are perhaps often induced, they are of such small dimensions, and offer so little obstruction to the blood-current, that their dissolution rather than their accretion and organization usually quickly results. The slight amelioration in symptoms, though promptly occurring, is of most ephemeral duration. Occasionally a "cure" is reported, yet this is so exceptional that the method must now be looked upon as scarce worthy of further trial.[3]

It must be briefly inquired, if the method originated and first practised by Moore, of London, that of permanent introduction of filiform material into the sac, which acts merely

[1] See paper by Powell "On the Diagnosis and Treatment of Aneurism of the Aorta," Lancet, January 4, 1890.

[2] Practised as advocated in text-books on electricity the treatment is useless indeed. A recent work—Liebig and Rohé's—recommends a current strength of but twenty to thirty milliampères, and this applied for but ten minutes. The inter-polar resistance is so great with one electrode externally, that an electrolytic action to any purpose necessitates a current strength and the duration of its application much in excess of this. Fifty milliampères are none too great nor an hour too long, if permanent results are to be hoped for. No greater risk attends the application of a strong current and a long session, than a weaker current and a short session, provided the needles are properly insulated. See my own case, in which a current of seventy milliampères was passed through two needles and two and a half feet of wire for an hour entirely without ill result.

[3] As to these reported *cures*, are they permanent? Unfortunately too few trouble themselves to obtain information as to later developments in rare cases of "cure" in other affections beside aneurism. The report is too often made *ad captandum;* the wonder excited is the reward desired.

mechanically, by offering multiple surfaces for clotting and by impeding the blood-stream, permitting the latter to whip itself about the former, is more promising than that of galvano-puncture. That it is theoretically so, there is no doubt, the two factors most important in cure being here furnished. It is not surprising, therefore, that since Moore, in 1864, attempted, by the method which has since been called by his name, to delay the fatal issue in a hopeless case of thoracic aortic aneurism, that the possibilities this treatment suggests should have caused it to be viewed with favor. It is, on the other hand, more curious that an interval of seven years should have been permitted to elapse before the method was tried a second time, possessing the theoretical advantages that it does. The cause probably lay in the subsequent unfortunate result in Moore's case—inflammation of the sac and pyæmia—one that in those days seemed inherent in the operation, but which was, in truth, rather due to the bad technique, faulty in the excessive amount of wire used,[1] with which were also probably introduced septic germs.

Of the twelve recorded cases in which wire was inserted,[2] without the conjoint employment of electrolysis, all were apparently in a more or less hopeless condition prior to operation. Yet, notwithstanding this, as regards clot-formation, reduction in the bulk of the sac, and its final obliteration, if such may be termed a cure, it would appear that two recoveries resulted, and the condition of several other of the cases was temporarily improved. In Loreta's case[3] an abdominal aneu-

[1] Twenty-six yards of fine iron wire were inserted—sufficient to originate much local disturbance. The wire was soft and bent in all directions (see remarks by Holmes and Hulke: Cayley's paper on a case treated with wire, Lancet, February 27, 1886). The aneurism was very large and seemed on the point of bursting through the skin or into the pericardium. Rapid coagulation in the sac occurred and temporary arrest of pulsation. Death resulted on the fifth day. (Medico-Chirur. Trans., xlvii. p. 129.)

[2] Omitting those in which watch-springs were used, the method of Bacelli, which has nothing to recommend it, either in theory or result, over the employment of ordinary wire. The elasticity of watch-springs necessarily interferes with subsequent contraction of the sac and its contents, without which an absolute cure cannot result. Their brittleness also renders fracture likely during process of sac-contraction. The walls of the aneurism are then endangered from contact with the sharp pieces.

[3] British Medical Journal, 1885.

rism, the size of a fœtal head at term contracted to the dimensions of a walnut, with obliteration of the sac cavity, by the seventieth day after the introduction of two yards of silvered copper wire. Death resulted subsequently from rupture of the aorta below the sac. In Morse's case[1] it is stated that eight weeks after the insertion of one and a half yards of one-half millimetre silver-plated copper wire into the sac of an abdominal aneurism the size of two fists, all indications of aneurism had disappeared, a hard nodule replacing the pulsating tumor. The patient remained well. In Cayley's case[2] of rapidly increasing thoracic aortic aneurism on the verge of rupture, forty feet of steel wire caused complete consolidation of that portion of the sac pointing externally, without constitutional disturbance or local pain. Two months later nearly thirty-five additional feet of wire were passed into the intra-thoracic portion of the aneurism, which now showed signs of rapid increase. Death resulted from dyspnœa from the tracheal pressure, without any change occurring in the symptoms or physical signs. The size and connections of the sac were stated by Cayley to have rendered the second operation ineffectual. That part of the sac in which the wire was first introduced was found to have undergone complete solidification. In the cases of Domville,[3] Murray,[4] Pringle (and Morris),[5] Lange,[6] and Ransohoff,[7] in which death followed the operation in a short time, the necropsies showed undoubted evidence of benefit having resulted from the introduction of wire. In Hulke's case[8] (thirty-three feet of wire) no *intra-vitam* or *post-mortem* change attributable to the use of wire could be noted. In that of White and Gould, in which thirty-two feet of steel

[1] Pacific Medical and Surgical Journal, February, 1887, abstracted in the Medical News, March 5, 1887. This operation was performed after Loreta's method, the aneurism being exposed through an abdominal section.

[2] Lancet, February 27, 1886.

[3] Stimson's Reference Handbook, vol. i.

[4] British Medical Journal, 1872, vol. i.

[5] Lancet, April 16, 1887.

[6] British Medical Journal, November 20, 1886.

[7] Medical News, May 29, 1886.

[8] Lancet, April 16, 1887.

wire were introduced, harm rather than benefit resulted, due, it was thought probable, to the use of an excessive amount of wire, and to too firm pressure being subsequently applied to the sac.

Closely following the second and third operation with wire, came a modification of Moore's method by Levis, that of the introduction of horsehair instead of wire. Subsequently catgut was used by Murray, of New Castle; and Florence silk by Schrötter, of Vienna. It was supposed that with these substances a nidus for deposit of fibrin would be offered with less risk of subsequent irritation, and with a better opportunity for contraction of organized thrombi and sac wall. It is not easy now to form an estimation as to the exact utility of these agents so used as methods of cure, based on the earlier operations, done as they were before the days of clean surgery. There can be little doubt that the ill result attending several of these was due, as in Moore's case, largely to septic complications. However this may be, the results with horsehair, silk, and gut have not been so encouraging, under like conditions, as those with wire, and this may be due to the fact that material of this sort can scarcely offer so suitable a framework as wire for the formation and support of coagula. It is not easy to fill a sac of some dimensions with such material as silk or gut, which is far more likely than the stiffer wire to be deflected to one side in its introduction by loose coagula present in more or less amount in all cases of old aneurism, so that its equal distribution about the sac is difficult or impossible. For this reason, if coagulation does not at once occur about it, less obstruction would be offered to the flow of blood in the sac, and thus an important factor in cure be lacking. But it is not for these reasons that I would advocate the superiority of wire. Despite the proved occasional utility of horsehair and silk, as in the cases of Bryant and Schrötter, in both of which firm laminated coagula engaged these substances, indicating that coagulation had been favored by their presence in the respective sacs; despite the innocuousness of such material when introduced with the strictest antisepsis, its susceptibility of admitting of the utmost contraction of clot and sac wall

without danger of subsequent irritation, it is not to be preferred to wire for the reason that all the objections which have been urged against the latter may be obviated by the use of a fine and but moderately drawn silver wire, which will permit compression into quite as small a bulk as horsehair or gut, with as little risk of subsequent irritation, and which, in addition, possesses the extraordinary advantage that a method can be conjoined with it whereby certain and prompt fibrin-formation will be promoted in the sac cavity. When it is considered that in many of the cases in which wire was used without electrolysis, especially the earlier ones, which were, of course, done without attention to antisepsis, far more wire was employed than was essential or harmless,[1] and that in many the operation was undertaken as a forlorn hope, the results obtained indicate that the method is not without utility. When practised in a manner that experience has taught us is free from risk and most likely to be beneficial, and then, also, combined with electrolysis, by aid of which firm coagulation in the sac can surely be effected, it would appear a most promising mode of treating aortic aneurism not yielding to the Tufnell method or to potassium iodide. With the anode as the active pole, the coagulum produced by galvano-puncture is much firmer than that which forms about unelectrified smooth wire; yet even with many needles in an ordinary-sized sac, as Barwell[2] points out, several trifling nodules of fibrin occurring at the periphery of the sac can have little effect on a large mass of circulating blood. With the combined method the chief objection to the use of a limited amount of fine aseptic wire disappears. Instead of a soft, unstable coagulum about the wire, tardy in appearing, there may be produced almost immediately a tough clot, which, in favorable cases, should tend by accretion to produce prompt obliteration of the sac cavity.

But seven cases of the combined operation are reported; these with my own, the eighth, are as follows:

[1] In Murray's case, twenty-four feet; in Cayley's, at first forty feet, subsequently thirty-five feet; in Hulke's, thirty-three feet; in White and Gould's, thirty-two feet.

[2] British Medical Journal, June 5, 1888.

Case I.—Burresi's (Corradi's).[1] Male, aged forty-three years; large aneurism ascending part aortic arch, non-responsive to carefully applied and varied medical treatment, or to the endermic application of electricity (method of Vizioli-Gallozzi). Case not thought to be propitious for any treatment, as sac-wall much thinned, communicating opening large, and tumor rapidly growing. A consultation resulted in the adoption of a method of treatment proposed by Corradi;[2] the latter inserted a canulated needle (diameter 8 mm.) into the aneurism in the second intercostal space, in a direction almost horizontal from left to right, and from before backward, penetrating the sac a distance of 2 cm.; 42 cm. (17 inches) of No. 30 annealed wire were passed. During its introduction the needle was circumducted with the object of winding the wire into coils on its entrance into the sac. The needle was then withdrawn and the extremity of the wire without the sac connected with the anode of a galvanic battery of sixteen elements, tested by a voltmeter to yield 1 c.c. of hydrogen gas for the first minute. Cathode to chest-wall. Current passed for twenty-five minutes. At the end of the first fifteen minutes of its application all pulsations had disappeared except that communicated from the adjacent part of the aorta. Operation was well borne. Pain entirely gone at end of third day. Patient continued to do well for a time, but subsequently all symptoms returned. Death at end of three and a quarter months.[3] No necropsy.

Case II.—Barwell's.[4] Male, aged thirty-nine years; luetic history. Very large progressive aneurism of the ascending and transverse part of the aortic arch; advanced pressure symptoms; medical treatment of no avail. When almost at the point of death, ten feet of the finest steel wire, which had been spirally wound, were passed through an ivory needle into one division of the sac. Anode active pole; cathode upon upper dorsal region; ten milliampères passed for one and one-sixth hours. Redness of skin produced at site of negative pole; no irritation at point of puncture. Signs of consolidation appeared at expiration of twelve hours: pulsation more distant; tumor firmer; pressure symptoms much diminished. On the fourth day of operation rapid increase in size of a secondary sac to the right of the former. Death on the seventh day from exhaustion and rupture of the secondary sac. Necropsy showed pronounced pressure effects on lungs.

[1] Lo Sperimentale, April, 1879. p. 445 et seq.; and also Giorn. Internaz. della Sci. Med., 1881, p. 1109 et seq.

[2] To Corradi is due the credit of first proposing the combination of galvanism with introduced wire, and of treating a case by the combined method. Barwell later, apparently unaware of Corradi's case, suggested a treatment on similar lines, though with a much superior technique to the latter.

[3] It is interesting to note that this case was reported *cured*, in Sperimentale, twenty-five days after the operation. The subsequent account of it is briefly given in a paper by Marcacci in Giorn. Internaz. della Sci. Med., 1881, p. 1109 et seq.

[4] Barwell: Loc. cit.

Primary sac contained much thick, firm, decolorized fibrin intimately united to sac and wire.

Case III.—Roosevelt's.[1] Male, aged twenty-five years; luetic history. "Aggravated aortic aneurism threatening death," involving upper four ribs on the right of sternum. Despite medical treatment, rapid advancement. Through a short, insulated aspirator-needle 225 feet of fine steel piano-wire (No. 00) were passed, connected with "one pole of constant battery" (which not stated); the other over right shoulder. "About twenty-five milliampères were passed for a half-hour." Potassium iodide (begun before operation) continued. Tumor pulsated less strongly the second day, pain and vertigo present. Third day, tumor less painful, but still pulsated; breathing "not so comfortable." Fourth day, dyspnœa and cyanosis. Seventh day, less pain and cyanosis; in better condition than before operation. During the third week could swallow and breathe with greater ease; tumor felt firmer. In fourth week vomiting and headache; potassium iodide discontinued. On twenty-second day "painful dark-colored spot appeared on one toe." Death on the twenty-third day. Necropsy not permitted.

Case IV.—Abbe's.[2] Male, aged forty-six years; no lues. Rapidly advancing aneurism at root of neck; oval cavity four inches by five inches; medical treatment unavailing; ligation of carotid, and shoulder amputation decided against. Case adjudged utterly hopeless. Barwell's operation resorted to for the purpose of lengthening life and to lessen pain; 100 feet of No. 1 aseptic catgut were introduced a few days before the wire, following which a part of the tumor seemed firmer, but rapid advancement occurred in other portions with pronounced pressure symptoms. On the ninth day following the use of catgut, 150 feet of fine sterilized wire were introduced through insulated aspirator-needle. Anode at first the active pole; cathode to back; fifty milliampères for one half-hour; one hundred milliampères second half-hour, cathode active pole, anode to back. No pain; pulsation continued, though tumor wall firmer. On second day rupture of the sac into trachea; pronounced pressure symptoms on trachea had long preceded rupture. Necropsy not permitted.

Case V.—Kerr's.[3] Male, aged thirty-eight years; luetic history. *Fusiform* aneurism from the base of heart to origin of left subclavian artery. Pressure symptoms. Medical treatment continued three months without result. Six feet of drawn silver wire were introduced through medium-sized hypodermic-needle insulated with shellac. Anode the active pole, cathode to epigastrium; current passed fifty minutes, its strength or the number of cells employed not stated. Impossible to force cut end of wire into sac through canula, canula therefore withdrawn, wire cut close to skin and forced in. Pain and pulsation stated to have been greatly relieved, though

[1] Medical News, April 9, 1887.
[2] Ibid.
[3] Occidental Medical Times, January, 1889.

death occurred on the eighteenth day. Necropsy revealed fusiform aneurism, as above. Wire had entered anterior surface of the sac. About the wire, as well as on the sac-wall, a firm clot had formed.

Case VI.—Kerr's.[1] Male, aged fifty-six years. "Aortic intra-pericardial aneurism," forming pulsating tumor on the right of sternum extending from the second to the fourth intercostal space. Pressure symptoms. No improvement on medical treatment. Electrolysis tried; two insulated needles in sac, anode active; negative on epigastrium; current passed for one hour. No improvement followed. Electrolysis through wire then used, *modus operandi* as above; ten feet of drawn silver wire were introduced; current passed for a half-hour, its strength or number of cells not stated. Within two months the patient left hospital "feeling as well as ever." Promised to report should symptoms return; no word at date of publication of paper.

Case VII.—Rosenstein's.[3] Obese young male, aged twenty-five years. Venereal and alcoholic excesses. Aneurism of the ascending part of the aortic arch, unimproved by the Tufnell-Balfour treatment. Pronounced pressure symptoms. Galvano-puncture tried; single needle; anode active; seventy milliampères for twenty minutes. Thirteen days later this procedure was repeated with two needles. No improvement. Five and a half weeks later, through exploratory trocar for six minutes a slowly increasing current up to seventy milliampères was passed; lance of trocar removed and two and one-sixth feet of spirally wound, moderately thick, softened silver wire, such as is used in trachelorrhaphy (about No. 28), were passed through the canula. The wire was pushed entirely into the sac with the lance, and the current applied for thirty minutes. Pain subsided in a few days; breathing became easier; tumor grew gradually smaller and harder, and the pulsations less. Pulsations had disappeared in the seventh week, complete recovery following.[4]

The history of my case, not before published, involving a number of points of interest, is related somewhat in detail.

Case VIII.—F. D. F., American, white male, of spare build, height about five feet seven inches; best weight about 140 pounds; occupation, insurance

[1] Loc. cit.

[2] Dr. Kerr informs me by letter that he lost sight of this patient about one year following the operation, and that all later inquiries regarding him have been fruitless.

[3] American Journal of the Medical Sciences, January, 1891.

[4] Under date of February 15, 1892, two years following the operation, Dr. Rosenstein informs me that his patient has continued cured. That he has had "no return of symptoms, except that in March last, after a comparatively mild though prolonged indulgence *in Baccho*, there was a slight irregularity of the pulse and a very faint bruit. Energetic measures as to total abstinence, more rest, and a little potassium odide with digitalis, helped him in about three months, and he has remained well ever since."

agent. First seen in July, 1888, then aged thirty years. About two years prior to this date pain had developed in the back in consequence of attempting to carry a heavy load. Lumbar pain thus induced, though slight at first, became severe after three or four days, and so continued steadily for about a month. Subsequently pain was chiefly localized in the left lumbar region anteriorly and posteriorly. Besides a dull, more or less continuous ache, there were lancinating pains in the area of distribution of the upper lumbar nerves. Several months before he was first seen, at a time when the pains were very slight or were absent, he indulged in violent wrestling, an exercise of which he was fond. Immediately afterward, while seated, he felt something give way in the former painful area. Shortly subsequent to this a small pulsating tumor appeared in the upper part of the left lumbar region. There was no history of leues. He was a trifle given to excessive venery, but was otherwise temperate. The bowels were much constipated. Examination revealed a small, expansile, pulsating swelling to the left of the upper lumbar vertebræ. Over it the second sound of the heart was plainly distinguishable, but no murmur. The latter was present somewhat above, at the border of the twelfth rib. There was no pulsation or murmur to be heard anteriorly. The heart was overacting, but otherwise normal. No difference could be dectected in the femoral pulses. Aneurism of the abdominal aorta was diagnosticated, and the patient received into the wards of the Jefferson Hospital. He was put at rest in recumbency on a spare diet, after the method of Tufnell. Potassium iodide was prescribed at first in doses of gr. x., three times daily, and subsequently in larger amount. The bowels were moved daily by laxatives, and codeine ordered in moderate doses for the relief of pain. The patient could be persuaded to keep at rest only with difficulty, and after a three months' sojourn in the ward, during which the pain much ameliorated and for a time disappeared, though no marked alteration occurred in the aneurism, he left the hospital. As it was thought that rest in recumbency rather than potassium iodide had benefited him, barium chloride was prescribed three weeks before his departure, at first in doses of one-tenth of a grain, and after a few days one-sixth of a grain, with seemingly remarkable benefit in the symptoms. Under this drug (other conditions as before), pain—which, despite every care taken to improve his condition, began to be as severe as formerly—now almost disappeared, and the area of aneurismal impulse somewhat diminished. He could not be persuaded to continue longer in the hospital, as his means were limited, and he felt sufficiently improved to continue his work. He was not seen again until seven months later (May, 1889). He then stated there had been little or no constant pain in the interval, and that he had been able to continue his work without interruption. During this period he had taken the barium chloride in doses of from one-sixth to one-third of a grain. The tumor had not increased in size. In the erect posture the impulse was slight, detectable only by palpation; in recumbency, prone, it

was perceptible to the eye, but not nearly so extended as at first, and its sounds were more distant, indicating that partial solidification had occurred. The heart was somewhat irritable, with a relatively accentuated apical second sound. The pulse was: standing, 100; sitting, 96. The bowels, as before, were unmoved without purgatives. Barium chloride was now increased to one-half grain doses.

I subsequently saw him but four times up to October 26th: once in June and three times in October. During this period, contrary to my advice, he actively pursued his vocation, which necessitated a great deal of walking. When seen in the autumn the improvement had ceased to be maintained. Without my knowledge or advice he had increased the dose of barium chloride to three-fourths of a grain a month before I saw him, on October 17th. The area of bulging and impulse was then larger, and the heart was very irritable. Barium was now discontinued. He consented to rest for a few weeks, and pursue the Tufnell treatment; but, after a short time spent in bed, he returned to work, and was not seen again until September last. He then had been confined to the house for two months, being too much prostrated and the pain too severe to go about. During the interval of two years in which I had not seen him he had taken little or no care of himself. His physical condition had grown progressively worse. Despite this, he continued his employment quite steadily until within a few months of September. More or less gnawing pain was felt in the region of the aneurism, with paroxysmal attacks of left-sided abdominal pain. The bulging posteriorly had reached a length of five and a half inches; its greatest breadth four and a half inches. There were three large nodules upon it, far more prominent than are shown in the photograph. Bulging began opposite the spinous process of the ninth dorsal vertebra, and extended to the lower lumbar spines. The abdomen and ribs were more prominent on the left side, which measured from mid-spine to mid-sternum one and three-fourths inches in excess of the right side. A marked impulse was perceptible to the eye and by palpation in all parts of the aneurism, the walls of which seemed thin and but ill-protected by clot. A murmur was heard over the most prominent portions. No sounds were detected laterally or in front over the prominent left abdomen. The area of splenic dulness was considerably increased. The heart was irritable, its sounds high pitched, the first lacking in muscular element; the impulse was slightly lowered and displaced to the left. There was a markedly accentuated pulmonary second sound, without any sign of an obstructive cardiac affection. The pulse was somewhat irregular and compressible. There were also cough, muco-purulent expectoration, and some dyspnœa. Mucous râles were present in the upper part of both lungs, accompanied by diminished resonance on percussion at the left apex. There was an area of anæsthesia extending over the aneurism from about the ninth dorsal spine above and the second lumbar below laterally and anteriorly toward the median line, in a direction slightly

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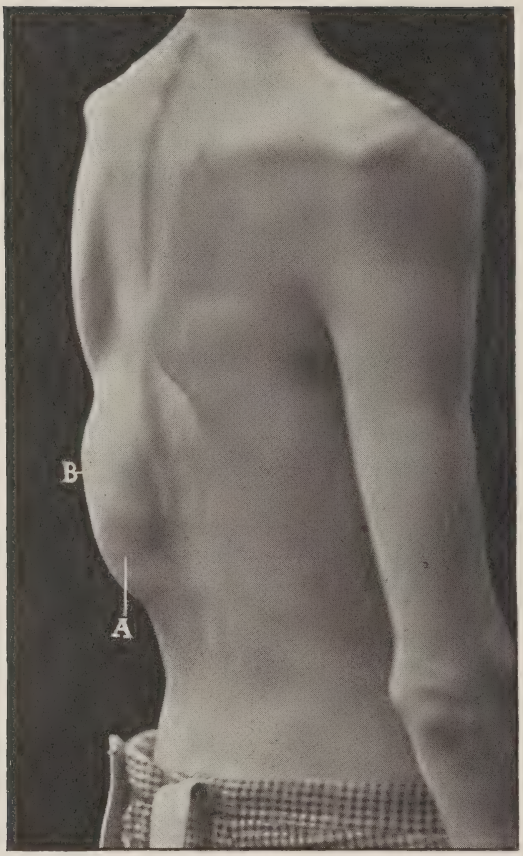

A. Point and direction of entrance of canula through which wire was passed.

B. Point and direction of entrance of platinum needle.

downward. Above and below this there was intensely heightened tactile and pain sense, the slightest touch or firm pressure causing much suffering. The superficial reflexes of dorsum, chest, and abdomen on the left side were extraordinarily heightened, very trifling stimulation producing markedly increased response on the same and the opposite side. The right knee-jerk was +, the left + +. Ankle clonus was absent. There was no paraplegia. The pain felt in the back was quite constant, lasting for hours, and was gnawing in character. Excruciatingly severe pain was felt in the left hypochondrium, loin, and abdomen. To relieve this he had been taking codeine in the extraordinary doses of gr. xx. to gr. xxx. daily. This was ordered discontinued, small doses of denarcotized opium at short intervals replacing it. He was removed to St. Mary's Hospital in September.

During his sojourn there his physical condition improved somewhat, but the area of bulging seemed to grow larger almost daily. As the physical signs indicated rapid advancement, and that rupture of the sac was imminent, the performance of electrolysis through introduced wire was suggested to him as offering a chance of at least slightly delaying the fatal issue, and, perhaps, also promoting euthanasia. He was told a cure was now impossible. With this understanding he was anxious for the operation. It was done on December 7th last, with the assistance of Dr. Pottberg, to whose mechanical skill I am indebted for many suggestions of value in the technique. Mr. Otto Flemming, who contributed the electrical outfit and needles, was also present. As my prime object in this case—in which a cure could not be expected—was rather to promote prompt formation of firm protecting coagula, in order to retard rupture of the thin-walled sac, than to cure an aneurism of such dimensions, with symptoms indicating advanced implication of vital parts, I chose a rather heavy silver wire;[1] for, on consideration, I concluded a better chance of immediate success lay in the introduction of wire of sufficient calibre to form large, supporting spirals in the sac, thus occupying considerable of its cavity, and affording a framework for clot, than in the use of a thinner, more pliant wire, which, though better calculated to permit of ultimate contraction of organized fibrin and sac walls—apparently not to be hoped for here—might undergo deflection from its course in introduction through impingement on loose coagula already in the sac, thus perhaps rendering it necessary to repeat the process, which from the patient's enfeebled condition would have been impracticable. A canulated steel needle, two and a half inches in length, of sufficient size to permit the passage of the wire, insulated with shellac to within half an inch of its point, was used to pass the wire. The wire and needles

[1] "Drawn hard to No. 23, Brown and Sharp gauge." It was so drawn that it would readily form spiral coils. It was afterward kept for several days on a roll two and a half inches in diameter, so that when removed and passed through a canula it assumed separate spirals each of about three to five inches in diameter, a few feet of it covering quite a large area.

were sterilized by boiling in carbolated water. The hot water somewhat softened the insulation, which was subsequently hardened in cool air. I feared to use carbolized glycerin to lubricate the needles before introduction, lest this also would disturb the insulation. The use of unsterilized glycerin or oil was considered unsafe because of the danger of sepsis, so the needles were passed after being simply remoistened in water. The canulated needle carrying the wire was inserted to within half an inch of its hilt. A platinum needle was also used, which was passed until it was thought that the insulated part was fully within the sac. I had primarily intended introducing the canulated needle in the upper, most prominent, and apparently the thinnest part of the sac wall, in a downward direction, as nearly as possible parallel with the long axis of the body, hoping in this way to favor coagulation that would protect the exterior wall, but little guarded by clot. Mr. Flemming had made for me four platinum needles of good calibre, two inches in length and insulated to the extent of one inch These I had also intended introducing below and over the most prominent parts of the sac, in a direction perpendicular to that of the canulated one, connected with the same rheophore, thinking that I might thus be able to secure the formation of multiple coagula extending from the wire to the needles and sac wall, and thus nearly consolidate the sac. Unfortunately for my purpose, it was found impossible to insert the canulated needle above in the direction desired, there still remaining some portions of the ribs yet unabsorbed, which interfered with its introduction. A trial was made in two places, but so much pain and disturbance of the sac being caused by my effort I desisted, passing the needle below, in an upward direction with the patient reclining prone.

Two and a half inches of wire were now slowly inserted, the distal end of which was first blunted by filing. An effort was now made to introduce the platinum needles. These, however, because of their cutting ends not being spear- or lancet-shaped, could be passed only with the greatest difficulty, the use of oil not being feasible, so that but one of the four was entered. The current was gradually increased through the controller until seventy milliampères were reached, and was maintained at this strength for one hour. The negative (indifferent) electrode was a very large felt plate, which lay upon the right shoulder and scapula, and was kept constantly moist with hot water. Notwithstanding this, and also that its situation was altered as much as space would permit, the large surface covered was the seat of intense pain, so that the current was momentarily opened through the rheostat to place a large folded, thoroughly-wet towel beneath the plate —a method I had before found to be of service when much pain is experienced from the contact of the felt when very strong currents are employed. Pain was unnoticed here after this, and none was experienced at the site of the active pole in either needles or sac. The pulse became quite feeble during the early part of the electrical treatment, but this weakness was not

maintained, the patient standing the ordeal well. Considerable difficulty was encountered in withdrawing the needle, due evidently to the firmness of the clot about them. I preferred not to practise a reversal of the current before their withdrawal, as has been recommended in the electrolysis of aneurism, lest the coagulum forming about the anode be softened by cathodic action. By gentle and continuous rotation and slight traction, with lateral pressure on the sac walls, they were removed, being also aided by an ingenious thought of Dr. Pottberg's, that of taking the lid of a stiff pasteboard pillbox, parting it from its periphery to its centre, and slipping it about the needle's base. Pressure made upon this by the fingers instead of directly on the sac enabled more counter-force to be utilized in withdrawal. Before removing the needles, an attempt was made to push the portion of wire yet within the canula into the sac. This procedure does not commend itself to my judgment, as it appears that such a method must inevitably disturb the relation obtained by electrolysis between the wire, clot, and sac wall, loosening the coagulum if attached to the sac. But a small portion had been passed in when blood appeared at the canula's extremity, and at the same time the patient stated that he felt something moving within, the first sensation that had been noticed in the sac. The needle was now cautiously withdrawn in the manner stated, the wire cut close to the skin and gently pushed beneath the latter, after drawing the skin as a cover over the wire.[1] During the introduction of the canulated needle blood spurted on the needle entering the sac wall to about the depth of one-third to one-half of an inch, this indicating the extreme thinness of wall, covered by skin, fascia, and muscle, and supposed to be lined by a certain amount of clot. Hemorrhage ceased on passing the wire into the canula. None occurred on withdrawing the needles. Their site of exit was sealed with cotton soaked in iodoform collodion.

The condition of both needles on withdrawal showed unquestionable clot formation about them. Both insulated and uninsulated portions of the platinum one, as far as it had penetrated into the sac cavity, were covered with thick, white, tenacious fibrin, in appearance resembling white paint. The steel canulated one was heavily coated with clot, and its uninsulated portion was partly decomposed, a slight touch on the cutting extremity crumbling it beneath the finger. A few hours subsequent to the operation the sac felt warmer than before, indicating inflammatory reaction, and pulsation seemed much less expansile. Considerable pain was felt in the centre and left side of the abdomen, but no more than had been present before the electrolysis. This was relieved by the passage of flatus and feces. Small doses of morphine and chloral were ordered should the pain become unbearable without their administration, the utmost quiet in recumbency was enjoined, and but little fluid in the shape of drink or food permitted.

[1] The skin had been pushed to one side in introducing the needles in order to form a valve-like opening.

The patient was very uneasy, as he had been before the operation, though for several days pain was not so severe as formerly, but the same amount of opiate was taken. It was impossible to keep him at rest. He tossed about the bed constantly, and could not be restrained at times from walking about the room. Despite this a remarkable change was noticed in the condition of the aneurism when the latter was examined on the third day. The prominent pulsating portion through which the canulated needle had been passed had sunken to the level of the general aneurismal surface, and transmitted pulsation alone could be detected in it. The whole of the lower part of the sac felt much firmer, and was quite without pulsation, while the extreme upper part seemed to have undergone no change. Yet all parts of the trunk about the sac above and below, laterally and anteriorly, could now be handled lightly and with varying degrees of pressure without the slightest discomfort on the part of the patient, the heightened tactile- and pain-sensibility having entirely disappeared, as had likewise the much-increased superficial reflexes. The tendon reflexes in the legs were not again tested. For several days the pain seemed less. Little, if any, darting pains occurred, but the gnawing sensation was felt deeply in the abdomen, probably due to erosion of vertebræ, and after the fourth or fifth day it became as severe as before. At the end of the ninth day succeeding the operation, immediately after the patient had been tossing himself about on the bed from the prone to the supine position and had been thumping his left side with his fist—a practice he was addicted to when in pain, and which he insisted upon pursuing though aware of its danger—he suddenly called for a cuspidor that he might spit, and almost immediately a gush of blood came from his mouth, and in a few seconds he was dead.

A necropsy was made eight hours succeeding death. Body had been placed in dorsal position; it was still warm. Blood oozed from the mouth when turned upon the side. The former aneurismal bulging had disappeared. On opening the abdomen the sac was not discernible without displacing the stomach and intestines. On removal of the sternum the left lung appeared collapsed. The anterior surface of the left pleural cavity was filled with fluid and clotted blood. The right lung was normally distended with air. Pleural adhesions existed on both sides above, laterally, and posteriorly. The pericardium contained a normal amount of fluid. The heart was one and a half times the size of the closed fist. Left ventricle especially hypertrophied. The valves were normal. The ascending and transverse aorta was slightly dilated saccularly, and the seat of extensive atheromatous degeneration. The spleen was six inches in length, five inches in width, and one and a half inches in thickness. Its anterior surface was adherent to the capsule of the left lobe of the liver, two and a half inches of which extended across a good part of the spleen. The anterior and posterior upper margins of the spleen and about one-half of the upper

posterior portion were intimately bound to the aneurism which lay above it and tended to push the spleen downward and forward.

The sac extended from a point about opposite the right nipple obliquely downward and to the left, across the bodies of the ninth, tenth, and eleventh dorsal vertebræ to the lower edge of the twelfth rib on the left, filling the hypochondrium and a part of the lumbar region. It was twelve inches in its oblique measurement, four inches in transverse diameter above on the right, and six inches below on the left. The aneurism had arisen from the posterior and left lateral wall of the lower thoracic aorta, and subsequently had involved a portion of the abdominal aorta. It had advanced beneath the base of the left lung, and thence downward, the diaphragm forming a partial covering. The anterior wall of the aorta was plainly discernible crossing the sac, the orifice of which measured two inches in length. An effort was made to remove the sac entire without disturbance of its contents, and the left lung with it, into which rupture had occurred. This was found to be impossible without mutilation of the exterior of the body, forbidden by the relatives. The boundaries of the aneurism, including, as they did, vertebræ and the greater portion of the posterior part of the right trunk, prevented the enucleation of the thin-walled sac without coincident removal of these structures. The sac was, therefore, opened *in situ*. It was yet distended with about a quart of clot and some fluid blood. The site of rupture—a one and a half inch tear—was in the upper left portion, the rip extending into the base of the lung, and thence upward toward the posterior part of the apex of the superior lobe and through the visceral pleura. The wire lay in coils in the sac. The highest portion of the most superficial coil was about two and a half inches below the point of rupture. The distal extremity of the wire lay in the same situation, its point curved somewhat away from the ruptured portion of the sac. The wire was adherent below to the sac wall, but whether by clot or through its proximal portion not having completely entered the sac, I could not discover, as when an effort was made to ascertain this, the wire and coagula had become loosened in the cavity, through a second attempt being made to remove the sac. Firm clots existed in all portions of the aneurism, with softened ones of evident very recent origin. The wire was engaged in several large firm clots which were of so solid a texture that when examined in that part of the sac which was removed they could be separated from sac and wire only with some difficulty.

The bodies of the lower dorsal vertebræ were much eroded, the spinal cord protected only by its membranes and thin laminated fibrin, lying exposed in the sac. These vertebræ formed part of sac wall, as did also the inner surface of the posterior and lateral portions of the inferior ribs on the left. These ribs, in parts, had almost entirely disappeared. All portions of the periphery of the sac contained more or less white, laminated coagulum, some of which had undergone organization. The rent in the

sac extended through laminated clot and sac wall into the base of the lung, ploughing for itself a sinus of some size. The calibre of the greater portion of the transverse colon, and all its descending portion, was much narrowed. The other abdominal viscera were normal. There were cretaceous nodules at the apex of the left lung.

Although these cases were treated by a similar method, a glance at the histories indicates that their character is so diverse as regards susceptibility of cure, and the application of the method so varied, that it is impossible to draw deductions from the results as a whole as to the utility of the procedure. The latter can only be arrived at by individual survey of each.

The cases of Corradi (Burresi's), Barwell, Roosevelt, and Abbe were apparently hopeless prior to operation, as was my own. In these more than decided amelioration in the symptoms could not be expected. In the first case of Kerr's, that of fusiform aortic aneurism, this plan of treatment could, of course, be of slight avail. In the remaining two cases, those of Kerr's and Rosenstein's, the results were decidedly beneficial, in the latter's absolutely curative; in Kerr's the patient was lost sight of, but not until subjective symptoms had entirely disappeared. The fact that he promised to return should the symptoms recrudesce, tends at least to indicate that this case has also remained well.

In Corradi's case, the chances of cure by any method were most remote. The decided temporary benefit following the operation, indicating prompt clot formation, shows, however, the possibilities of this procedure, which, as practised by Corradi, was faulty in the small quantity of wire used, and in the fact that the wire was not spirally wound before introduction.

In Barwell's case, despite its unfavorable nature, undoubted signs of consolidation in the sac resulted, a fatal issue being due to rupture of a second sac into which the wire was not passed. The presence of firm decolorized fibrin adherent to the wire and sac illustrated the beneficial effects of the electrolysis. Although a current of much greater strength than ten milliampères would probably have been still more productive of good, the final result would have been, of course, similar.

In the case of Roosevelt the amount of wire used—two hundred and twenty-five feet—was probably much in excess of that necessary to fill the sac with coils and sufficient to interfere with subsequent contraction of the aneurism, had the case been susceptible of cure by this method. In Abbe's case one hundred feet of catgut had been introduced nine days prior to the insertion of one hundred and fifty feet of fine steel wire, a quantity greater than could be attended with the best ultimate results had a cure been within the range of probabilities. Here, too, the method was faulty, in that the polarity of the current was reversed after fifty milliampères had been passed by the anode for a half-hour; the effect of one hundred milliampères through the negative pole for the same time subsequently, probably being to soften and partly dissolve the firm coagulum formed about the positive pole.

In these four cases fine steel wire was used. In Kerr's cases and in Rosenstein's and my own case, silver wire was employed. Kerr used ten feet of silver wire of a calibre somewhat greater than the bore of a medium-sized hypodermic needle which it was drawn to fit. Rosenstein used two and one-sixth feet of softened silver wire. In my case, two and one-half feet of drawn wire of rather large calibre were inserted.

As the immediate cause of death in a number of cases in which filiform material was used has been rupture of the sac, it is important to inquire as to the influence of introduced substances, and especially wire, in its production. Rupture may follow the insertion of filiform material of any sort, in consequence of the partial obliteration of the sac cavity by formed coagula, resulting in rapidly raised pressure on an unprotected and weakened portion of the sac now exposed to a greater pressure than formerly. This was probably the cause of rupture in Barwell's case and my own, as well as in those of Domville, Ransohoff, and others,[1] in which a ne-

[1] In these, coagula forming about the wire had not completely filled the sac. In my case the sac was of such dimensions that the spirals did not reach much beyond the upper half, the wire having been introduced from below, and but two and one-half feet having been used.

cropsy showed that no wire lay near the site of rupture. With the use of too firm a wire, or of wire in too great quantity, rupture is especially to be feared as a result of it interfering mechanically with contraction of fibrin and sac wall, and tending by its resistance to weaken a part of the latter. If steel wire be used, an additional danger may lie in the likelihood of its fracture in several places during contraction of the consolidated aneurism. The sharp extremities of these broken portions might be productive of great damage to the sac wall. Rupture apparently is only likely from the direct action of the wire when unyielding material, such as steel or highly-drawn silver, is used, or when excessive amount of wire of any sort is introduced. Therefore, while it is important that the wire used should be sufficiently firm to be readily introduced, it must, on the contrary, not have so much permanent spring as to resist contraction of the clot of which it must necessarily form a part. Soft iron wire has been especially recommended by Steavenson,[1] as best fulfilling the various indications. He suggests that, during the passage of the current, soft iron wire would undergo decomposition with the formation of chloride and oxide of iron, which would in addition exercise their one specific coagulating property. I was at first much inclined to the use of soft iron wire in preference to wire of other metals, until I undertook some experiments as to the influence of currents on the former, the result of which has caused me to prefer a wire less likely to be decomposed by the battery. In passing a current of fifty milliampères for one hour's time through ten feet of No. 36 soft iron wire, connected with the positive pole, and placed in twenty ounces of three-quarter per cent. salt solution, the circuit closed by the cathode in the fluid, but not in contact with the anodal wire, an amount of detritus resulted, representing iron chloride and oxide, from the decomposition that had occurred between the wire and the solution under the influence of the current, that would be dangerous to release in an aneurismal sac. A portion of it would, of course, become engaged in the

[1] Lancet, July 11, 1887.

forming clot; another portion, however, might readily be carried into the circulation, and provoke the formation of thrombi elsewhere. A current of much less strength had a proportionately similar effect; that of ten milliampères for the same time also caused the formation of much sediment. Since it is of the utmost importance that a firm thrombus be promptly formed about the wire, and as this is more likely to result through the passage of a strong current for a considerable time, such as fifty to seventy milliampères for one hour, and since this strength of current for this time, carefully applied, is attended with no more risk than a less strength for a briefer time, it is important that current be strong and the session long. But this would be attended with the above-mentioned danger if soft iron wire were used, and as steel is also objectionable for the reason before stated, wire of another metal, such as silver, is to be preferred. Silver salts will not be formed under the current's influence in appreciable quantity, and yet the wire will be sufficiently corroded to favor the deposit of fibrin upon it. The wire should be fine, and should be drawn just sufficiently hard that it can be readily passed through the canula forming spirals in the sac of about the size desired.[1] The cases apparently in which the best results were obtained, both when wire alone was used and when rëenforced by galvanism, were those in which ten feet or less of wire were introduced.[2]

As Kerr remarks, six to ten feet are sufficient to introduce into any aortic aneurism, the object being merely to favor deposition of fibrin, and at the same time to provide a supporting matrix for the clot. As a means of demonstrating that but little wire is essential, he suggests the passage into a bottle

[1] Kerr (loc. cit.) directs that the calibre of the wire selected be somewhat larger than the diameter of the canula, and that it be drawn through a plate until it develops spring enough not to "kink." It must not, however, be overdrawn, as in such a case it will not coil away from the point of the canula, but will impinge on the opposite wall of the sac, as was the case in a third patient upon whom Kerr attempted the introduction of wire.

[2] Loretta's case, practically cured when death occurred, ten feet; Morse's case, cured, four and a half feet; Kerr's case, thought to be a cure, ten feet; Rosenstein's case, cured, two and one-sixth feet.

the size of the aneurism, through a perforated cork, of sufficient wire to fill it with loops. It then will be seen how few feet are necessary to come into contact with all parts of the bottle's interior. He believes, also, that when a greater amount than ten feet is employed, there is danger of the excess passing away in loops into other portions of the vessel, or, when the sac is in juxtaposition to the heart, of it entering the ventricle.

DISCUSSION.

Dr. Charles W. Dulles: I once made some observations on the introduction of horsehair into an aneurism of the aorta. In 1875 there was in the Philadelphia Hospital a celebrated case, a man with an enormous aneurism, the swelling of which had eroded the ribs and protruded over the chest of the right side so as to resemble the breast of a well-developed woman. The case was under Dr. Pepper's care when I was his resident in the medical ward. It was subsequently transferred to the late Dr. Maury's care in the surgical ward. Dr. Maury introduced a large quantity of horsehair into the sac; and for a time the symptoms of aneurism diminished. The tumor grew firmer and seemed to diminish in size, but before long it again increased and Dr. Maury ligated the right common carotid and the right subclavian arteries. This produced no material effect, and the aneurism subsequently ruptured. An autopsy was made, and one of my colleagues and I made a very careful dissection of the aneurismal tumor and the surrounding parts, and we found that there was considerable laminated clot on the walls of the sac. This surrounded and enclosed the horsehairs which had been introduced. It appeared that the effect of the introduction of the horsehair had been very favorable, and we had the impression that if the sac had been nearly filled this might have resulted in solidification of the contents of the entire cavity.

A few years ago I made somewhat of a study of the treatment of aneurism by the introduction of filiform material, and was favorably impressed with the effects of the introduction of wire with or without electricity. I have had no personal experience in the use of these materials, but I believe that if the use of horsehair were given a more extended trial the result would be satisfactory. The greatest misfortune about this matter is that the treatment described is not adopted until the case is almost hopeless; and no method which is used under such circumstances can be said to have had a fair trial.

Dr. Arthur V. Meigs: When I was resident in the Pennsylvania Hospital I had charge, under the late Dr. Levis, of a patient with an aneurism of the innominate artery, into which Dr. Levis introduced a large quantity of horsehair. The aneurism projected on the right side above and below the clavicle. The man subsequently died, and I was present at the post-mortem made by Dr. Longstreth. It was one of those cases in which there is no distinct wall. There was much disintegration of the wall of the sac, so that it was impossible to say where the aneurism ended and the lung began. A part of the horsehair was in the lung. The patient died quietly five or six weeks after the introduction of the horsehair. I do not see how any procedure can be expected to cure conditions of this kind, which are in their nature almost incurable because of the damage that has been done.

Dr. H. A. Hare: There is a great degree of variation in regard to the rapidity of the clotting of blood in the bloodvessels and the heart. The blood clots around different foreign bodies with different degrees of rapidity. Around a glass tube it clots slowly, even when ground or rough glass is used, but if a little piece of ligature enters the bloodvessel the clot occurs at once. I am not sure that this might not be an advantage in the use of horsehair, or perhaps thread or threads wrapped around wire. This might increase the clotting. I have never used a wire to produce clotting, but I have introduced a wire to remove a clot, and noticed that, under these circumstances, the clotting rapidly occurred. I have, however, attributed this to irritation of the endothelial lining of the bloodvessel rather than to any effect on the blood itself.

Dr. Henry F. Formad: I recall a case which gives us an idea of the rapidity with which clotting of blood may take place in a healthy individual. A man was stabbed with a knife in the heart and lived for twenty-five minutes. In the injury one of the columnæ carneæ in the left ventricle was cut, and around this cut column there was found a distinct white, firm blood-clot the size of a small walnut. This clot was white and strongly adherent to the severed column. There were, of course, the usual red clots besides, but it was evident that in twenty-five minutes this white, firm, ante-mortem clot had formed. The cut columnæ carneæ in this case acted like a foreign body around which the clot formed.

Dr. J. M. DaCosta (by the secretary) presented to the College:

A stethoscope formerly owned and used by Laënnec.

A clinical thermometer which once belonged to Wunderlich.

A clinical thermometer used by Cullen.

THE DIFFERENTIAL DIAGNOSIS OF RUBEOLA AND RUBELLA; WITH ESPECIAL REFERENCE TO THE ENLARGEMENT OF THE GLANDS OF THE NECK.

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[Read April 6, 1892.]

Before beginning the subject proper of this paper, I wish to say a word about the term "rubella." I find that many physicians do not seem to understand exactly what is meant by this. I do not wish to be regarded as crowding upon the profession any new name. This word is not new. It was advocated in 1866 by Veale as a title for what is commonly called German measles. It has been adopted by many other writers. For example, it is used by Squire, and is the reference word in Quain's *Dictionary of Medicine*. Ashby and Wright employ it as one of two titles. It is used also as the title of the section in the *Cyclopædia of the Diseases of Children*, and in the new *Textbook of Medicine* by Osler. Atkinson, too, adopted it in his valuable paper upon this disease, which appeared in 1887. If we do not use this term we have to fall back upon "rötheln," which, as Fagge truly says, is a "barbarous expedient." There is no more reason for applying "*rötheln*" to German measles than for calling ordinary measles *masern*. Both are purely German words, and the former is unpronounceable by the untrained English tongue. Moreover, rubella expresses the relation of the disease to measles just as varicella expresses the relation of that affection to variola; both rubella and varicella

being distinct diseases with slight symptoms resembling in many particulars the other two graver affections respectively.

In spite of the light which has been thrown upon the diagnosis of these two affections by writers of later years, the recognition of either in certain cases is even yet by no means always an easy task. So difficult, in fact, is it sometimes, that there probably are many who still doubt that rubella exists as an independent affection. Yet the majority of those who have seen much of the two diseases, or at least who have studied them carefully, now believe that they are quite distinct from each other.

It would be an easy matter to quote numbers of reported instances to prove that the occurrence of either rubeola or rubella does in nowise give immunity from the other disease. Out of nearly 60 cases of rubeola which I treated in 1887 in an epidemic in the Southern Home for Destitute Children of this city, 7 cases had had rubella under my observation in a large house-epidemic which occurred the year previously. One constantly meets with children who are said to have had measles twice, though all the highest authorities admit that it is very rare for the disease to occur twice in the same individual. There is no question that most of these cases are instances of the development at different times of each disease, neither of which protected from the other.

The difficulty in diagnosis consists in the fact that the symptoms of rubella vary greatly within certain limits. Particularly does this apply to the eruption, which is, to a certain extent, multiform. As I have already stated elsewhere,[1] we may meet, in reality, two forms of the affection, so far as the rash is concerned—a *rubella morbilliforme*, which has an eruption almost identical with that of measles, and a *rubella scarlatiniforme*, in which the rash is so like that of scarlatina that only the subsequent course of the case may clear up the diagnosis. These two forms are, however, *not* distinct species, as Filatow has contended, but merely varieties; and gradations of every degree may be observed between the two extremes.

[1] N. Y. Medical Record, July 2d and 9th, 1887.

As instances of the two forms, the following cases may be of interest. Both patients were inmates of the Home referred to, and were sick in the same room at the same time, together with a number of others who exhibited rubella in its typical form. An epidemic of rubella was prevailing in Philadelphia at the time, and no case of measles or of scarlatina was met with in the institution that year.

Case I.—Bertha L., aged eight years.

March 6, 1886, 2 P.M. Without previous symptoms an eruption appeared this morning, accompanied by injection of the conjunctivæ. The rash is now very abundant, very slightly elevated, widely spread, consisting of macules of small pin-head size, or confluent in large patches. In some places it presents a uniform red appearance. The face is almost a solid red, but, on close inspection, shows in this a red mottling or "maculation." The glands below the angle of the jaw, behind the lobe of the right ear, and behind both sterno-cleido-mastoids are enlarged. The palms and both the palmar and dorsal surfaces of the fingers exhibit the eruption. The tonsils and anterior pillars of the pharynx are red; the posterior wall of the pharynx slightly so. No eruption can be seen in the throat. The tongue is not coated. The child coughs and sneezes, though not to a great extent. Pulse, 147; temperature, 102.4° F.

7*th*. The face is deeply flushed. The rash is everywhere confluent over the body, not at all elevated, and presents the greatest possible resemblance to that of scarlatina. Only in a few localities, viz., the wrists, the edges of the soles, and the forehead, does careful search reveal a few discrete, pin-head-sized macules. The cough continues. Pulse, 129; temperature, 102.7° F.

8*th*. The eruption has faded considerably, and is only slightly visible on the face, and not at all on the arms and trunk. On the legs it is almost confluent, but shows small pinhead-sized macules on very close examination. Temperature, 99.6° F. Yesterday evening the patient complained of pain on swallowing, but to-day makes no complaint, although the pillars of the pharynx and the tonsils are bright red. The glands behind the ear and the sterno-mastoids are still enlarged, but those below the jaw are no longer so.

9*th*. The eruption has disappeared except from the nates. Pale yellowish spots are visible on the face, with some branny desquamation. The skin of the arms is dry throughout. The throat presents a nearly normal appearance.

10*th*. Desquamation of fine scales is very visible on the limbs and trunk, but not on the palms. Temperature, 99.6° F.

11*th*. The gland behind the ear is no longer enlarged, but those behind the sterno-mastoids are still so. Temperature, 99.8° F. Desquamation continues.

There is no question, I think, but that the great majority of observers would have pronounced this case to be one of scarlatina had it occurred singly, particularly if it had been seen for the first time in the condition which it exhibited on March 7th. I must freely confess that I have made this mistake in other cases similar to this, in which the patient was seen in private practice outside of local epidemics.

Case II.—Katie G., aged five years.

March 4, 1886, 11.30 A.M. Yesterday evening the child's eyes were congested and watering. As these symptoms continued this morning she was kept in bed. At breakfast-time a rash was observed on the face. At the present hour the face is covered with pea-sized blotches more or less confluent. A very few are visible on the trunk and extremities. The skin of the face, and to some extent of the body as well, is harsh to the touch. There is marked lachrymation, photophobia, and injection of the conjunctivæ. The glands under the angle of the jaw and behind the sterno-cleido mastoids are enlarged, but there is no enlargement behind the ear. Temperature, 103.8° F.; pulse, 153. The tonsils are swollen and red and the pharynx red. The child twitches considerably, and is excessively nervous in all its movements. A hot bath and a febrifuge ordered.

5*th*. Improvement in the nervous symptoms took place at about 4 P.M yesterday. The eyes improved and the child grew brighter. The rash was still limited almost entirely to the face. It has now spread all over the body, is composed of quite elevated, deep purple-red maculo-papules, which exhibit a marked tendency to grouping in lines and in decidedly crescentic patches. The eruption is in most parts of the body almost or quite identical with that of measles. On the back, however, the papules are quite small and of a brownish-red color, and there is not so great a tendency to grouping as elsewhere on the body. The face to-day has much less rash upon it, and the patches there are fading and indistinct. Temperature, 100.3° F.

6*th*. The eruption has left the face and is fading elsewhere. It is now best marked in pea-sized and smaller papules at the ankles and wrists. Temperature, 99.6° F.

7*th*. A branny desquamation is beginning in the face and arms, and indistinctly elsewhere as well. There is no cough. The tongue is clean. The glands are still swollen. The throat is but slightly red. Temperature, 99.7° F.

Had this patient been met with as an isolated case the diagnosis of measles could have been made with great propriety. Compared with many mild cases of measles which all physicians see so frequently it may be said that this case actually "out-measled measles."

These instances are detailed as illustrations of the difficulty sometimes attending the diagnosis of rubella. Although a careful consideration would show that even in these cases a diagnosis is possible from the symptoms as detailed, yet it is manifestly unfair to select atypical cases of any disorder in which to search for diagnostic symptoms.

What then are the principal factors on which to base a diagnosis between rubeola and rubella?

The period of *incubation* of rubeola is perhaps most often stated as fourteen days. This seems to be a figure fairly well fixed, though subject to some variation.

Rubella has an incubation period of a considerable range, as evidenced by the various statements given by different writers. One to three weeks perhaps best expresses the truth of the case. But little aid, therefore, is to be obtained from a comparison of the incubation periods of the two diseases.

The mode of *invasion* is usually quite different in the two affections. The prodromal symptoms of rubeola last three days, as a rule. There are coryza, cough, lachrymation, and photophobia. Often there are symptoms of croup. The temperature is considerably elevated. The child is commonly sleepy and stupid—"sleeping for the measles," as it has often been expressed. Vomiting is not infrequent. Sore-throat is not usually complained of. The whole group of symptoms clearly show that the little patient is quite sick.

In rubella, on the other hand, the prodromal symptoms are usually either entirely unnoticed or last but twelve to twenty-four hours, and are of little import. They resemble the catarrhal symptoms of measles but are of slighter grade. Vomiting, however, is rare, while sore-throat is very common. Fever is absent or slight. Drowsiness is a very frequent symptom in my experience.

When we compare the *eruptive* stages of the two diseases the diagnosis becomes easy in typical cases. In rubeola we find a distinct, papular, dark-red or purplish-red rash, which appears first on the face on the fourth or fifth day of the disease. These papules have, as is well known, a peculiar disposition to

arrange themselves in groups, forming straight or curved lines; the individual papules then becoming confluent to some extent. The rash spreads slowly, taking one to three days to cover the whole body, and still persisting in full blow on the face while this extension is progressing. Thus the maximum intensity over the whole body is reached by the second or third day of the eruption. The eruption may also be present in the oral cavity and on the pharynx. After the maximum intensity is reached the rash begins to fade, first from the face and later from the rest of the body. With the development of the eruption there occurs an increase in the severity of all the symptoms already mentioned among the prodromes. Diarrhœa is not uncommon and severe bronchitis is frequent. As the rash disappears these symptoms decline rapidly in intensity, except that bronchitis is very liable to persist for a time.

Rubella, on the other hand, presents an eruption which, as already stated, tends to vary greatly. Ordinarily it consists of pale-rose, indistinct maculo-papules. These are usually only slightly elevated, and are decidedly smaller than those seen in measles, for although they vary in size from that of a pin-head to that of a split-pea, the majority are of the smaller dimensions. They have none of the duskiness or purplish hue of the rash of measles. They are, for the most part, discrete, closely placed, and widely diffused. When confluent, they are so in large, irregular patches with little or no disposition to form small groups of a crescentic shape. The method of the spreading of the eruption over the body is interesting. One of two courses may be observed. In the first the rash is often present all over the body when first discovered, reaches its maximum everywhere by the second day, and then fades rapidly. In fully as many cases, however, a characteristic sequence can be observed. The rash then appears first on the face and spreads rapidly to other parts of the body, reaches the hands and feet last of all, and begins to fade from the face and trunk, or even disappears entirely from them, before it appears at all, or before its maximum intensity is attained, at the extremities. Thus it passes over the body like a wave, reaching its maxi-

mum on any one part within twelve to twenty-four hours after its appearance there; and usually beginning to fade from the last part involved within twenty-four hours after its first appearance anywhere. The eruption is sometimes found in the throat, though probably not so often as in measles.

The symptoms attending the eruption of rubella are slight suffusion of the eyes, a tendency to coryza and sneezing, a slight cough and moderate elevation of temperature. Although the catarrhal symptoms may occasionally be quite severe, yet in the great majority of cases they are either insignificant or are absent entirely. Sore-throat, however, is a very common and characteristic symptom. Sometimes it is slight, but in many cases under my observation it was marked, and swallowing was painful. Nearly all writers agree regarding the frequency of the occurrence of sore-throat in this disease. In twenty-four hours from the development of the rash the symptoms usually begin to improve rapidly. A branny desquamation not infrequently follows, though this is less commonly observed than in measles.

This description of the diagnostic symptoms applies to typical cases of the two diseases. Unfortunately, many atypical cases of rubella resemble either scarlatina or rubeola, especially the latter. And conversely, cases of measles may be so light and exhibit so poorly defined an eruption and such slight constitutional symptoms that they may strikingly resemble rubella; while the same statement is true of mild, anomalous cases of scarlatina. Errors of diagnosis are common and will continue so, for it is sometimes absolutely impossible in sporadic cases to recognize with certainty any one of these three disorders.

There is one symptom of which I have as yet made no mention, but which has long been regarded by many as almost pathognomonic of rubella, viz., the enlargement of the superficial cervical and posterior auricular glands. Could we depend upon this as a characteristic symptom of rubella, we should be in possession of a diagnostic feature of great value. A number of writers maintain that this enlargement is one of

the prodromal symptoms. I have never had the opportunity of examining for it before the rash had appeared, but I have rarely failed to discover it during the eruptive stage. In fact there is no doubt whatever that the enlargement during this stage is very widely regarded as—as it in reality is—one of the most constant symptoms of the disease. Yet the fact, though long known, seems largely to have been forgotten, that a similar enlargement may occur in measles. It is certainly safe to say that most practitioners do not look upon enlargement of the superficial cervical and posterior auricular glands as at all a prominent symptom of measles, and probably that few ever examine for it except in doubtful cases.

As far as is indicated by a study of a number of the leading text-books on medicine and on diseases of children, and of the journal literature for the last five years, the majority of writers either make no reference at all to glandular enlargement in rubeola, or speak of it in only the most general way. A few, however, do call attention to the matter. For instance, Hardaway[1] refers to the swelling of the glands in measles, although he certainly attaches especial importance to the symptom in rubella. Squire[2] likewise, although mentioning the enlargement in measles, emphasizes the value of the symptom in rubella. The text-books of Gerhardt and of Vogel-Biedert contain references to a widespread enlargement of lymphatic glands in rubeola, and Waxham[3] says that enlargement of the submaxillary and anterior cervical glands is common. It is somewhat doubtful what group of glands this writer intends to designate by "anterior cervical," but he can hardly have the superficial cervical group in mind. A word of caution regarding diagnosis comes from Atkinson,[4] who speaks of the constant occurrence of glandular enlargement in rubella, but says that "it is likewise very often observed in measles." Townsend[5]

[1] System of Medicine by American Authors.
[2] Quain's Dictionary of Medicine.
[3] Cyclopædia of the Diseases of Children.
[4] Reference Handbook of the Medical Sciences.
[5] Boston Medical and Surgical Journal, August 22, 1890.

says that enlarged cervical glands are sometimes found in rubeola and sometimes not in rubella.

Finally, Swift,[1] although speaking of the cervical adenitis as almost pathognomonic of rubella, yet states that in an epidemic of measles under his observation in 1886, 24 out of 27 cases exhibited a glandular enlargement exactly similar to it. He refers to a published statement of my own[2] to the effect that I had not infrequently found this enlargement in measles.

Since publishing this statement I have continued to be greatly interested in studying measles with especial reference to this question. Of many of the cases which I have seen I have unfortunately preserved no record, and even many of the records taken were hastily made and necessarily brief and imperfect. There are, nevertheless, 37 cases of rubeola in which there is recorded an enlargement of the superficial cervical glands, and 11 in which the posterior auricular glands were involved. Of these latter cases 8 belong to the first category as well. In the remaining 3 no note was made of the condition of the superficial cervical group. There are a large number of other records in which the condition of the glands is not mentioned, and which are consequently useless for the present purpose. I find, however, that only in 2 instances do my notes positively state that no enlargement was present in the glands in question.

It is true that the swelling in the cases of rubeola was usually not so marked as in rubella. It was, however, quite decided enough to nullify very greatly, for me, at least, the diagnostic value of this symptom.

It has been an interesting observation also, to which I can only allude here, to discover enlargement of the superficial cervical glands in several cases of scarlatina, in at least 3 of which the diagnosis from rubella was made with some difficulty, although the subsequent peeling settled the question beyond cavil.

Inasmuch as it is conceivable that very many children might

[1] Reference Handbook of the Medical Sciences.

[2] Medical Record, 1887, July 2d and 9th.

possibly exhibit a chronic enlargement of the superficial cervical and posterior auricular glands, and that the presence of this symptom in measles might be an accident only, I have examined a large number of children who were either apparently healthy or were suffering from no eruptive fever, but have found the enlargement in only very few instances, unless the children were very markedly strumous. The conclusion, therefore, seems justifiable that, although this special glandular enlargement is a very constant symptom in rubella, it is probably nearly equally as frequent in rubeola, and that it possesses by no means so great diagnostic importance as is usually supposed.

Note.—Since writing the above I have seen 13 additional cases of rubeola, in all of which the superficial cervical glands were enlarged.

DISCUSSION.

Dr. Arthur V. Meigs: There is a point to which I think Dr. Griffith did not allude, and it is the fact that in Philadelphia the belief is prevalent among the public that second attacks of measles are common. My own belief is that this is due to the failure to distinguish between German and real measles—two affections which are now, and have been during the last ten years, quite common in this community.

My own experience coincides with that of Dr. Griffith in regard to the post-auricular glands—their enlargement will not materially aid in making a differential diagnosis.

Dr. Thomas D. Dunn, of West Chester: There is one point that has assisted me in distinguishing between the two affections—that is, the eruption in the roof of the mouth. In rubeola it is marked in the early stages of the disease. It is seldom present in rubella. My attention was first called to this point eight years ago by Dr. Jacob Price, of West Chester, and I have depended upon it in many cases, not only in distinguishing between the two affections, but as the earliest positive diagnostic sign of rubeola, it being present from twenty-four to thirty-six hours before the eruption on the face.

I had some two years ago an unusual opportunity of verifying a statement of Dr. Griffith. In the West Chester State Normal School there are present under one roof four or five hundred students. During an outbreak of

rubeola in that institution there were a good many cases of rubella in the town. There were about thirty-eight genuine cases of measles in the school, and of this number, in the course of six or eight weeks, eight took rubella. This clearly shows that the one disease does not protect against the other.

Dr. J. Madison Taylor: I had it in my mind to say just what Dr. Dunn has so well said. The punctation of the vault of the pharynx is one of the most important early diagnostic signs of measles. At my dispensary at the Children's Hospital, very frequently we are enabled to predict the occurrence of measles from twelve to twenty-four hours, or even sometimes longer, before the eruption breaks out elsewhere; and the capacity to foretell thus, even before any other specific signs are evidenced, is of extreme value, not only in a dispensary service where epidemics can be systematically noted and suspected cases watched, but it is especially valuable in large institutions where many children are grouped together. I know of few text-books where this sign is described. It was first called to my attention by Dr. Louis Starr many years ago, and I am surprised that it has not received more widespread attention. It belongs peculiarly to rubeola, but is occasionally seen in rubella.

I also agree with Dr. Meigs, that many of the cases of what we are apt to describe as second attacks of measles are clearly instances of rubella. I have two cases now under my observation, puzzling me very much at first, but which I am sure are of this latter disorder. An important distinguishing characteristic in the eruption of rubella is a roughened, harsh quality of the skin, noticeable on touch or when viewed in profile. In the two children, sisters, referred to, the first went through an attack almost unrecognized by the mother, and I only saw it when called upon to minister to the second one, who was quite sick. At that time the first was desquamating, in fine branny scales. The second child had coryzal symptoms, pharyngitis, severe cervical adenitis, and desquamated finally in large plates. The eruption was dusky and coarse, much elevated above the surface of the skin, and quite unlike any scarlatina I have ever seen. The desquamation of the rubella is, I think, very irregular in character; sometimes almost absent, sometimes branny, and not seldom shed in flakes, making one feel very suspicious of scarlatina. I have in my hand to show you several colored drawings, I made just ten years ago, of some very severe cases of malignant measles seen at the Philadelphia Hospital. In these you will see the tendency to crescents clearly noticeable. I think in most cases of true measles this crescentiform eruption can always be recognized at some period. In rubella it is not so.

Dr. Walter D. Green: I do not know that I have anything more to add except in the way of corroboration. Five years ago there was an outbreak of measles in Girard College when I was resident physician there under the late Dr. Reed. There were four hundred and ten cases, and I think the percentage of cases in which the eruption appeared in the pharynx

was something like eighty-eight. Dr. Reed laid a great deal of stress upon this as a diagnostic point.

Dr. Griffith: I did refer to the matter of second attack to which Dr. Meigs has alluded, and my opinion agrees with his.

Though it would be impossible, I presume, to convince the laity of the rarity of the occurrence of measles twice in the same person, yet as physicians we must accept all these statements with the greatest suspicion and unbelief.

The eruption of measles in the throat and oral cavity is to a certain extent a diagnostic sign, it is true. I have sometimes, however, seen it well marked in rubella, and this has been the experience of a number of observers. It would not be safe, therefore, to base a diagnosis of measles on this symptom alone. In all these diseases, in fact, one has to be guided by the general complexus of symptoms. We cannot choose any one symptom.

I have only briefly referred to the great similarity to scarlatina sometimes seen, because I hope at a future day to make some statements in regard to that. In one of our best text-books on medicine the writer says that rubella can scarcely be confounded with scarlet fever; but I am sure that many of us will agree that we should accept such a statement with a great deal of caution. I recollect well how, just after I had published a paper on rubella, some years ago, and thought I knew something about the recognition of the disease, I was called to see two children in one family. One had little, if any fever, was not sick at all, and had an indistinct rash which was more like that of rubella than like anything else. I pronounced it rubella. The baby also had a slight rash on the inner surface of the thighs, and I told the mother that I could not believe that that was more than a "baby rash" due to heat or indigestion. In a few days both children were peeling in a very characteristic way. One of my earliest cases in practice was a little girl with acute scarlatinous nephritis. The child had been but slightly ill. The diagnosis of rubella had been made by another physician and no precautions taken. Certainly, in every case where there is the slightest doubt, it is not only safe but it is our duty to treat the patient as though scarlatina were present.

POST-DIPHTHERITIC PARALYSIS OF THE EXTERNAL RECTI; WITH A CASE.

By G. E. de SCHWEINITZ, M.D.,

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[Read April 6, 1892.]

Paralysis of accommodation, or cycloplegia, without alteration of the condition of the iris (mydriasis) is often seen after diphtheria, and is the most constant affection of the internal ocular muscles that occurs as a sequel of this disease. Usually bilateral, the cycloplegia is rarely complete, but when it is there may be mydriasis. There is, however, considerable diversity of expression concerning the state of the pupil under these circumstances. Sphincter palsy has been recorded by Weber[1] and by Donders[2]; sphincter paresis by Schaeby-Buch,[3] and Abercrombie[4] found the pupil dilated and sluggish in all of eighteen cases. Other observers regard the affection of the pupil as comparatively rare, and for the most part are in accord that there is generally an absence of pupil-symptoms.

Just as palsy and paresis of the ciliary muscle are frequent, paralysis of the external ocular muscles is comparatively uncommon. Faure,[5] among other visual disturbances, mentions

[1] Virchow's Archiv, Bd. xxv. and xxviii.

[2] Accommodation and Refraction of the Eye. New Sydenham Society's Translations, 1864, p. 297.

[3] Archiv f. Ophthalmologie, xvii., p. 265.

[4] Trans. International Medical Congress, 1881.

[5] L'Union Médicale, Nos. 15 and 16, 1857; quoted by Donders, loc. cit.

strabismus once. Among ninety cases recorded by Maingault,[1] amblyopia was present in thirty-nine and strabismus in ten. Rumpp[2] speaks of paralysis of the internal recti muscles. Alfred Graefe[3] noted two cases, in one of which there was paralysis of the superior oblique, and in the other of the external recti. Remak,[4] among one hundred cases of post-diphtheritic cycloplegia, found ten with paralysis of one or both external recti. Rosenmayer,[5] among ten cases of post-diphtheritic cycloplegia, observed two with paresis of both external recti. Arthur H. Benson[6] records a case in which the levatores palpebrarum were affected in the ninth week after the throat lesion appeared, and during the same week the external recti were attacked, and there occurred convergent strabismus and diplopia. Gowers,[7] speaking of the affections of the special senses in diphtheria, refers to weakness of the internal recti, which may be associated with the cycloplegia, and quotes Vadelot as authority for one case in which all the muscles supplied by one third nerve were paralyzed. He has seen double ptosis, and also mentions the occasional phenomenon of slight paralysis of one or another of the ocular muscles, varying from day to day, and Pagenstecher[8] has observed in an epidemic of diphtheria, cases of palsy of the external ocular muscles (recti and obliqui) characterized by rapid onset, interchange and disappearance, occurring in the convalescent stage. Recently A. Stanford Morton[9] has reported three cases of more or less complete bilateral paralysis of the external recti following diphtheria.

Several cases of paralysis of all of the ocular muscles after diphtheria have been published, but this complication is exceedingly uncommon. Thus Uhthoff[10] records the case of a

[1] Nagel's Jahresbericht, 1871. [2] Ibid., 1877, p. 381.
[3] Graef. u Saemisch's Handbuch der gesammten Augenheilkunde, vi, p. 73.
[4] Centralblatt f. prakt. Augenheilkunde, 1886.
[5] Wien med. Wochenschr., No. 13, 1886.
[6] Trans. Oph. Soc. of the U. K., 1883, vol. iii., p. 265.
[7] Diseases of the Nervous System, 1888, p. 1223.
[8] Monatsbl. f. Augenheilkunde, Jahrgang 64, p. 358; quoted by A. Graefe, loc. cit.
[9] Trans. of the Ophthalmological Soc. of the U. K., 1891, vol. xi., p. 106.
[10] Neurolog. Centralbl., 1885, No. 6, p. 125.

boy, aged ten years, who had been treated for a mild attack of diphtheria for sixteen days. Some time later he was unable to read. Nineteen days after recovery from the diphtheria there was paralysis of accommodation, and ten days afterward complete external ophthalmoplegia. Mendel[1] describes a case which terminated fatally in a boy aged eight years. Diphtheria of moderate severity was present for a week. Six days later there was paralysis of the palate; about a month later visual disturbance and weakness of all four extremities; and three days after this, double ptosis, most marked on the right side, paresis of the external rectus, paralysis of the internal rectus, with paresis of the superior and inferior recti. In the left eye there was paresis of all of the recti muscles. There was no paralysis of accommodation or limitation of the visual field. Ten days after the onset of the ocular paralysis, the patient died. Evetsky[2] has published the case of a girl, aged eight years, who had complete bilateral ptosis, abolition of the upward and downward movements of the eyes, limited lateral movement, no loss of accommodation, and normal visual fields and color-sense. Two weeks previously the child had had a sore throat, with painful deglutition.

To the cases of paralysis of the external rectus muscle which have been recorded, I desire to add the following example, which occurred in the Infirmary for Nervous Diseases, in the service of Dr. Weir Mitchell, by whose kind permission I publish it:

F. B., aged four and one-half years, was brought for treatment January 8, 1892. The following record was made by Dr. Joseph Leidy: The patient, when four months of age, had an illness which was diagnosticated as spinal meningitis, the diagnosis being based upon the following symptoms: Retracted head, bulging on the top, and a cephalic cry, with fever. There was no permanent lesion after this attack. Six weeks before the date of her visit to the Infirmary the child contracted diphtheria, from which she made a slow recovery. Three weeks after the diphtheritic attack, the following symptoms were noted: Ptosis, irregularity of the pupils, the right being larger than the left, and weakness of the right and left external recti muscles.

[1] Ibid., 1885, No. 6, p. 128.

[2] Arch. d'Ophthal., November and December, 1887.

The child regurgitated liquid food; there was difficulty in swallowing; the speech was indistinct; hearing and taste were unimpaired; there was tremor of the hands and arms, and muscular weakness of the extremities. She is now unable to walk or stand, or at least indisposed to attempt to do this; knee-jerk absent; no ankle clonus; no incoördination of the hands. The child is nervous, irritable, and cries out frequently during sleep. The appetite is fair, the tongue clean, the bowels regular. The lungs are normal. There is paralysis of the palate, as before noted, liquid foods being regurgitated. The heart's action is rapid, the pulse 120, but there is no murmur.

I made an examination of the eyes, with the following result: There is convergent strabismus, the right eye "fixing"; the lateral movements are diminished, the left eye failing to move past the median line to the left, and the right failing to pass to the internal commissure. The vertical movements are unimpaired. Attempts to demonstrate diplopia were not satisfactory, but it probably was present, and, so far as I could judge, there was false projection of the field of vision. The pupils were unequal. Facing a bright light the right pupil measured 5 mm. and the left pupil 3 mm.; their reaction to light and to convergence was unimpaired. There was slight ptosis of the left eye, the upper lid upon that side drooping a little lower than that of the right, causing a narrowing of the left palpebral fissure of one-sixteenth of an inch. Rough tests seemed to show that there was paralysis of the ciliary muscles. The suggestion of Dr. Edward Jackson to employ the shadow-test to elicit this point was thought of, but it was impracticable to utilize it on account of the extreme restlessness and irritability of the child; indeed, at this time all of the examinations were very difficult. Ophthalmoscopically there was no change of importance. Each optic disc was a vertical oval, rather pallid, and the retinal veins a little fuller than normal.

The patient was ordered an elixir of quinine, iron, and strychnine, faradism for the lower extremities, massage and rest in bed.

Three days later, *i. e.*, on January 15, 1892, she was admitted to the hospital, and the ocular conditions were then as follows: Right pupil 4 mm., left pupil 3 mm.; ptosis as before; the right eye now moves to the external commissure; in the left eye the limitation of movement remains; there is still slight convergent strabismus and the child fixes by preference with the right eye; the pupils react normally.

The child was placed in bed, a pill of strychnine sulphate, of gr. $\frac{1}{100}$, three times a day was ordered, together with general massage and faradic electricity. The following notes made by the house-doctor, Dr. Dean, are taken from Dr. Mitchell's case-book.

January 25, 1892. No regurgitation of liquid; no difficulty in swallowing; appetite good; speech much improved and words easily distinguished; no tremor of the arms and hands; disposition much improved; rarely cries, and is generally smiling and ready for conversation; cannot walk without

support, being unable to place the feet in proper position; when in the recumbent posture, however, there is good control of the movements in all directions. She cannot overcome much force with the lower extremities. The hand-grasp is of fair strength and equal on both sides. Knee-jerk absent; tendo Achillis-jerk absent; plantar reflexes present; no clonus; elbow-jerk absent.

March 10. Walks without assistance but gait faltering and unsteady; speech as good as other children at her age; appetite good; muscle-power much improved in lower extremities; power of resistance in arms and forearms good; knee-jerk absent on both sides; reinforcement absent; tendo Achillis-jerk absent; plantar reflexes present; no clonus; elbow-jerk absent; no chin-jerk.

11*th*. I made the following record in regard to the eyes: Strabismus entirely absent; excursion of the eyes normal in all directions; pupils still slightly unequal, but their reactions normal in all particulars. The strabismus disappeared about a week after admission.

The last examination (March 18th) which has been made of this child revealed the following points: No strabismus; excursion of the eyeballs normal in all directions; width of the palpebral fissures equal; slight inequality of the pupils, the right being one-quarter of a millimeter larger than the left; muscular power good and walks easily; knee-jerk, elbow-jerk, and tendo Achillis-jerk still absent.

There are several points of interest in this case.

1. The order in which the paralytic symptoms appeared: It is difficult to state this accurately, but it seems likely that the throat affection lasted for about three weeks, that the soft palate, and probably at the same time the ciliary muscle, were affected in the fourth week, followed immediately by the ptosis or affection of the levator palpebrarum. The strabismus is also placed in the same period; while somewhat later, probably in the sixth week, the weakness of the lower extremities began. In Benson's case the primary throat affection lasted four weeks, the ciliary muscles were affected in the fifth week, the soft palate in the sixth week, the hearing in the sixth week, the levatores papebrarum and the external recti in the ninth week, and the lower extremities in the tenth week.

2. The order in which recovery occurred: First the accommodation returned, and almost at the same time the weakness of the right external rectus muscle disappeared, that is, there

was no difficulty in conjugate movement to the right; later, and within a week, the limitation of the movement to the left had disappeared. In close connection with this was the return of the child's natural disposition, which was pleasant and lovable. Following this came improvement in the lower extremities, so that six weeks after treatment she was able to walk without assistance. The knee-jerk, as is usual in these cases, remains absent, and is probably the last of the functions to reappear. In connection with the order in which the ocular symptoms disappeared in this child, Uhthoff's case of complete paralysis of all of the ocular muscles is interesting. First the accommodation recovered; then the muscles engaged in certain associated movements, namely, the inferior recti and conjugate deviation to the left, that is, left external rectus and left internal rectus; then the superior recti recovered; and finally conjugate deviation to the right was possible.

3. The unilateral mydriasis: There was marked inequality of the pupils for more than a month, and even now, eleven weeks after the first pupillary phenomena were noted, there is slight difference in the diameters of the pupils. If it is true that mydriasis is more apt to occur when the cycloplegia is complete, it is probable that this may have been the condition on the right side, while on the left it was incomplete. At no time was there any imperfection in the motility of the irides, either under the stimulus of light or convergence.

Although this of necessity is purely a clinical record of bilateral diphtheritic palsy of the external recti and incidental mention of the accompanying cycloplegia, it is worth while in closing to refer briefly to our knowledge of the pathology of these cases. At the present time diphtheritic palsy is usually included among the acute nuclear palsies, but according to Dr. Gowers, although it is probable that diphtheritic cycloplegia is of nuclear origin, it is uncertain whether the paralysis of the orbital muscles, when it occurs, should be ascribed to nuclear or peripheral disease. The history of the theories which have been advanced to explain diphtheritic paralysis in

general is somewhat as follows: Charcot and Vulpian[1] attributed the palsy to an ascending neuritis from the local lesion. Buhl[2] ascribed the condition to vascular changes, capillary hemorrhages and exudations both in the brain and around the roots and ganglia of the spinal nerves. Déjerine[3] ascribed the cause of diphtheritic palsy to a subacute poliomyelitis involving the motor cells of the anterior horns and their homologous nuclei, with a secondary peripheral neuritis. Oertel and Klebs have found microörganisms. Mendel, whose case of complete ophthalmoplegia externa came to post-mortem examination, believed the condition due to capillary hemorrhages (central) and a primary peripheral neuritis. Siemerling[4] in a superb research concerning chronic progressive paralysis of the ocular muscles, concludes that the local lesions found in chronic ophthalmoplegia justify the conclusions, that there may be:

1. Nuclear disease (disappearance of the ganglion cells) with participation of the nerves to their termination in the muscles.

2. Degeneration of the muscles and of the nerve-trunks with intact nuclei.

3. Interruption of the conducting power of the intra-medullary roots on account of sclerotic foci, with intact muscles, nerve-trunks, and nuclei.

These propositions, of course, do not refer especially to diphtheritic cases, but are interesting in this connection on account of their authoritative statement in regard to the pathological condition underlying progressive paralysis of the ocular muscles.

[1] Compt. rend. de la Soc. Biolog., 1862.

[2] Zeitschr. f. Biologie, 1867.

[3] Archiv de Physiologie Norm. et Patholog., 1878, p. 167.

[4] Archiv f. Psychiatrie und Nervenkrankheiten, 1891, Bd xxii., suppl. Heft.

DISCUSSION.

Dr. Edward Jackson: I have been interested in this matter of post-diphtheritic paralysis ever since I suffered from it myself a year after I engaged in the practice of medicine. While the affection is quite rare, I believe that it is not so uncommon as the literature would seem to indicate. I can now recall five cases that I have seen where the extra-ocular muscles were affected. And, curiously enough, two of these have occurred in my service at the Polyclinic since the first of the year.

One of these was a boy, thirteen years old, who came for double vision, three weeks after the onset of the diphtheria. The diplopia was homonymous and the deviation was quite perceptible objectively, and the images separated more widely to the right or left of the visual field than at the centre, showing a marked weakness of both external recti muscles. There was also a vertical deviation from paresis of the left superior rectus. There was no perceptible weakness of accommodation or change in the pupils. In two weeks the diplopia had disappeared when the patient looked directly in front, but appeared on looking twenty degrees to the right or left. The defect of vertical movement was gone. Shortly after this the recovery was complete in all respects. I think that recovery occurred within six or seven weeks after the onset of diphtheria.

The second case was that of a girl, seven years of age, who had had diphtheria two months previously. She had recovered from the throat affection and had been sent back to school, and was brought because she could not do her school-work satisfactorily. In her case there was also homonymous diplopia, increased toward the limit of the field on either side, showing weakness of both external recti and the diplopia. The accommodation was reduced to 1 D. in each eye. This case was seen two weeks ago, and has failed to report subsequently.

These cases were very similar to the others that I have seen. One of the cases was under observation several months after the diphtheria had occurred with well-marked squint. Whether there was subsequent recovery or not, I do not know. All the others recovered. In the cases that I have seen the affection was not a complete paralysis. It is rather a paresis.

I fancy that we we do not see the mild cases, and the probability is that there are many mild cases to one severe one. In all the cases that I have seen there has been marked weakness of the affected muscles for some time before they came under observation. They have evidently tolerated a moderate amount of trouble for some time before they thought it worth while consulting anyone about it. Considering the early age at which diphtheria occurs and the character of the phenomena, it is probable that many cases escape notice that would be discovered if all cases of diphtheria were examined carefully with reference to the strength or weakness of the ocular muscles.

Dr. James Hendrie Lloyd: I was under the impression, until I heard the paper, that paralysis of some of the external ocular muscles was not rare in post-diphtheritic paralysis; but as it seems to be rarer than I supposed, it may be worth while to state briefly the details of a case under my observation two years ago. I attended this child, five years of age, through the primary disease. He made a good recovery from a bad attack of faucial diphtheria. Four weeks after my last visit the patient was brought to my office with paralysis of one external rectus muscle, paralysis of the velum palati, weakness of the legs and arms, abolished knee-jerks, complete head-drop, the muscles of the back of the neck being paralyzed and allowing the head to fall forward on the chest. I had the boy admitted to the Home for Crippled Children, where I had an opportunity to study the progress of the disease. The thoughts which that case gave rise to were with reference to the probably constant association of paralysis of the external eye muscles with paralysis of the velum palati; whether in doubtful cases the paralysis of the external eye muscles would be associated with involvement of the velum palati, and with cycloplegia, thus making a characteristic symptom-group which would help in making the diagnosis. It is well known that cases of post-diphtheritic paralysis are occasionally mistaken for other diseases, especially when the primary disease has been slight and its true nature overlooked. The establishment therefore by observation of such a symptom-group might be occasionally of great advantage.

Dr. J. P. Crozer Griffith: I know so little about diseases of the eye that I would hesitate to say anything in this connection were it not that I wish to ask for information. There is no doubt that text-books on diseases of children put paralysis of the ocular muscles as a quite frequent form of diphtheritic paralysis. Probably the most frequent form is absence of the knee-jerk, then paralysis of the palate, then, according to most writers, paralysis of some one or more of the ocular muscles. One of two things is certainly true, either the text-books are wrong or else many of these cases do not come to the ophthalmologists, but rather to the men who are treating cases in general medicine or diseases of children. I should like to know in what category the following two cases should be placed, of which I have here the very brief notes: A little girl, seven years of age, came to my clinic in the University Hospital this past winter. Six weeks previously she had been taken with diphtheria. She was sick two weeks, and then, when recovering, the mother noticed that the child could not walk well, and that her head was bent forward, with the chin on the breast. The speech was nasal, and in the effort to swallow, the food regurgitated through the nose. The child walked with a shuffling gait, with the head on the chest. The knee-jerk was absent on both sides. There was convergent strabismus, but the pupils reacted to light. Coördination was bad. The child staggered when the eyes were shut.

The second patient was a girl baby, fifteen months of age, who had diph-

theria three or four weeks previously. For two weeks she had been choking when nursing. The head dropped upon the chest, the child could no longer walk, and there was convergent strabismus.

This strabismus cannot be explained on the ground that it is due to general weakness, such as would occur after all febrile diseases. We do not see this symptom after other febrile diseases. To what is this convergent strabismus due? I do not report these cases as instances of paralysis of the external recti, for I do not know that that was the cause. I presume that paralysis of the muscles of accommodation might be a factor in producing strabismus, but it is about that point which I wish to be informed. It is, in any case, clear to me that paralysis of some ocular muscles existed in these cases. Although I could make no such careful examination as Dr. de Schweinitz did in his case, yet I do believe that these children were suffering from paralysis of the external recti, and that the paralysis differed only in degree, but not in kind, from that seen in the case reported by him. The same statement must apply also to the other cases reported here this evening, and to the statements made by the writers of our best text-books, who certainly have had extended experience in this matter. They cannot all be wrong. It must be that the ophthalmologists meet only the graver, more persistent cases, and it is perhaps to these that Dr. de Schweinitz refers, although he did not make any such distinction in his paper.

Dr. Charles Shaffner: In a large dispensary practice I have seen these cases of post-diphtheritic paralysis frequently. It usually occurs about a week or two after the patient gets out of bed. The patient soon observes that there is something wrong with the working of the muscular apparatus, usually the external muscles. Sometimes there is mydriasis, with interference with the accommodation, and the case often gets well if left to itself. It is regarded as a functional disease, similar to other paralyses, due to the circulation of a poison in the blood. Syphilis will sometimes do the same thing. The rheumatic poison will also cause paresis. There seems to be a state of the nerves easily acted upon by the poison, whatever kind it may be. I cannot explain its cause, although I know that it is a functional disease, and easily curable. I usually give these patients beef-tea, punch, and tincture of the chloride of iron. I have seen such cases get well in a few days.

Dr. Charles Seltzer: In the experience of the general practitioner I think that this is a common affection. I have seen two cases since the first of the year—one was extremely severe. In both, the eyes were convergent. As far as treatment is concerned, I find that instillation of atropine, paralyzing the accommodation, gives the greatest relief for a time. Recognizing that the affection disappears under the use of tonics, etc., temporary relief is best obtained by the use of a mydriatic.

Dr. Charles K. Mills: I have seen a large number of cases of post-diphtheritic paralysis, and my experience is that palsy of the ocular muscles

is comparatively rare. The cases that come to me, however, come usually some time after the attack of diphtheria, and it may be that the eye affection has been present and has disappeared. One of the first cases of diphtheritic paralysis which came under my observation was, however, one in which there was double ocular motor paralysis, associated with paralysis of all four limbs, and with slight facial paralysis; also with evidences of involvement of the muscles of the neck and possibly of the pneumogastric. The patient seemed to be in danger of death when I first saw him, and was apparently rescued only by active stimulating treatment, both local and general. Subsequently, in the course of five or six months, he recovered entirely. In a disease like diphtheria we would naturally expect to have any form of paralysis. The poison certainly may act both upon the nerve centres and the peripheral nerves anywhere. I have no doubt that the cases are frequently to be explained by a concurrent poliomyelitis and neuritis, due to a concurrent toxæmia of both the centres and the periphery. While this is not always easily proved, I have seen a few cases in which it was very evident that neuritis and poliomyelitis, or at least affections of the cornua, were present. Not a long time since I saw a patient with extreme hyperæsthesia and pain, and with paralytic disorders, and who eventually was left with permanent atrophic paralysis, of the ordinary form of spinal infantile paralysis. I have but little doubt as to the pathology of most cases, that they are usually nuclear affections.

Dr. Arthur V. Meigs: It would be hardly correct to say that diphtheritic paralysis is a common affection, but one of the commonest forms of it is some affection of the eye muscles. I can remember several instances in which I have seen internal squint as a result of diphtheria, coming on usually about six weeks after the attack. Listening to what has been said, it has struck me that the reason that no more attention has been bestowed upon this affection is that diphtheritic paralysis is most common in young children, and it is in young children that it would be less likely to be recognized and most difficult to study. The instances in which I have seen internal squint have all been in young children. I recall one case which I saw fifteen years ago. I have had a similar personal experience with that of Dr. Jackson. After I was grown up I had an attack of post-diphtheritic paralysis, with eye affection.

Dr. de Schweinitz: Of course, post-diphtheritic paralysis of the internal ocular muscles, especially of the ciliary muscle, is exceedingly common. With this type of palsy the paper has nothing to do, except in incidental mention. Paralysis of the external ocular muscles, the result of diphtheria, may be classified as follows:

First, as a quite common occurrence, there is temporary strabismus, usually convergent, such as most of the general practitioners here have seen, which rapidly disappears, and which usually occurs toward the convalescent stage. This, for example, was the case in the epidemic referred to and described by

Pagenstecher. With the ordinary cycloplegia there is quite commonly present weakness of the internal recti, to which I have merely referred in passing.

Second, there may be complete bilateral paralysis of the external recti, which is less common, and of which the case reported to-night is an example, although the right external rectus was probably not completely paralyzed.

Third, there are cases in which there is paralysis, not of the external ocular muscles themselves, but of certain associated movements. For instance, there may be failure of conjugate movement to the right or to the left. This seems to be a more uncommon condition.

Finally, there are the exceedingly rare cases in which there is complete external ophthalmoplegia, and of which there are only a very few cases on record. The one reported by Dr. Mills this evening makes an additional one. It seems to me that Dr. Mills expresses the correct view in regard to the lesions—a view somewhat confirmed by the post-mortem examination of Mendel's case. In many instances there is a peripheral neuritis, and this is sometimes associated with central changes.

A CASE OF GIANT GROWTH OF THE COLON CAUSING COPROSTASIS OR HABITUAL CONSTIPATION.

By HENRY F. FORMAD, M.D.,
DEMONSTRATOR OF MORBID ANATOMY AND LECTURER ON EXPERIMENTAL PATHOLOGY AT THE UNIVERSITY OF PENNSYLVANIA.

[Read April 6, 1892.]

The case to be recorded, and from which I present the specimens, is unique in the history of morbid anatomy. It represents a human colon of dimensions equal to that of a cow, and the accompanying illustration, which is a reproduction of a photograph of the specimen, taken side by side and simultaneously with a normal human colon, will convey to those who did not see the specimen an adequate idea as to its dimensions. (See Plates I., II., and III.)

The exact measurements of the different parts of the bowel will be given hereinafter.

Definition. The specimen is one of *giant growth* of the colon, or a congenital anomaly belonging under the category of *monster per excessum*, followed by coprostasis or habitual constipation, which eventually terminated fatally in the adult. This case differs from all on record in the following points:

1. In that the enlargement or dilatation preceded and was the cause, and not the result, of coprostasis.

2. There was no acquired morbid change, such as colitis or obstruction of the bowel, that led to this condition.

3. The mode of death was not due to ulceration or perforation of the bowel, or to peritonitis, as in the cases on record; and,

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PLATE I.

Human colon, congenital giant growth and coprostasis. The more distended end is the sigmoid flexure. The narrow part taking exit from it, represents the greater part of the rectum, which was normal. The narrower distal end of the preparation represents the head of the colon with the string attached to a fragment of the small intestines. The arched part of the specimen represents the transverse portion of the colon. The figure within represents a normal human colon photographed simultaneously for comparison of dimensions. Dried preparations.

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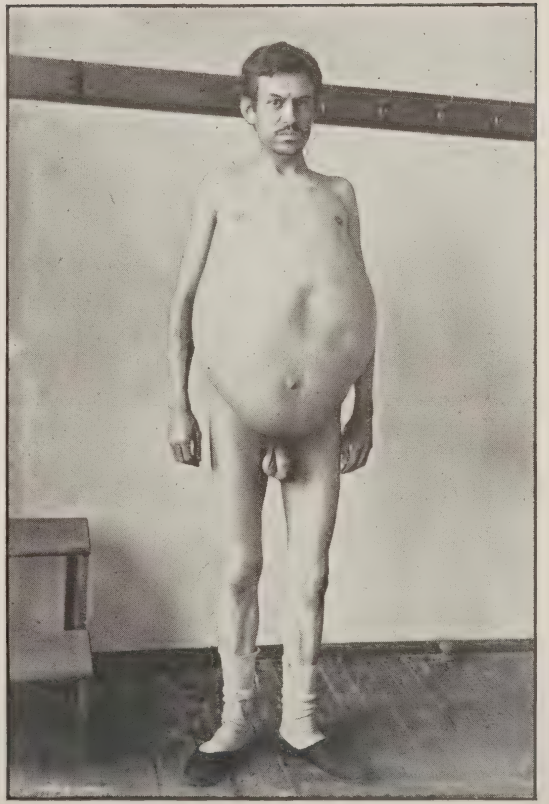
PLATE II.



Case of coprostasis, showing distention of abdomen.
Front view.

PLATE III.

Case of coprostasis, showing distention and shape of trunk.
Side view. [All three illustrations relate to the same case.]

4. The specimen excels in magnitude the few cases heretofore recorded.

History of case. J. W., aged twenty-nine years, white, single, was found dead in the water-closet on the premises of a society to which he belonged, and the case subsequently became subject to the investigation of the coroner. Being delegated to make an autopsy and an investigation of this case, my inquiries revealed the following facts:

His mother tells that up to the age of one and a half years the subject under consideration was a normal infant, with the exception of a rather large abdomen, frequent irregularity of bowels, and attacks of constipation, but no other deformity of the body had been noticed by her up to that time. Subsequently, and especially noticeable at the age of two years, the abdomen began to swell and the disturbances in defecation began to be more marked, so that constipation would last from two to four days as a rule. His appearance was said to be that of a marasmic child, lean and emaciated, and until five years of age he was unable to rise without assistance. Subsequently the bodily development was progressive, although he was rather lean. At the age of twelve years he was able to go to school, and although the history of his intellectual success as a schoolboy is uncertain, he appeared to have had the normal intelligence of lads of his age. At the age of sixteen years he earned his living at a foundry for eighteen months at continuous work. Subsequently he worked for several years as a laborer at an oil refinery, and while his work was uninterrupted during this period, his parents say that he was subject to habitual constipation, said to last as long as a whole month at a time, although no correct clinical data covering this period of his life could be obtained. Yet it was obvious that his abdomen continued to grow in size. At this period of his life, while not feeling distressed by any painful ailment, he is said to have visited the dispensaries of various hospitals of this city. At twenty years of age his abdomen had reached very large dimensions, and the figure of his body became so peculiar that the managers of the Ninth and Arch Streets museum saw fit to put him on exhibition as a "freak;" and for eight or ten years he was known as the "Wind-bag" or "Balloon man." It is said that at this time the circumference of his body was equal to his height, and that he was able to diminish or increase the calibre of his abdomen and chest at will to the extent of fourteen inches. He took particular delight in pounding himself upon the abdomen with his fist, and permitted spectators at the museum to do likewise. Eye-witnesses say that when his abdomen was thus pounded it resounded like a drum. It appears evident that his abdomen must have been larger at previous periods of his life than at the time of death. For about one year he ceased appearing as a museum freak and acted as a messenger for a market, being quite a well-known figure on the streets during the year previous to his death.

The whole history of his life and habits does not present anything peculiar, except that he had an enormous appetite and was generally a good feeder. He was known to relish a few extra heavy meals a day. He was occasionally of intemperate habits.

He applied lately to the Hahnemann Hospital, complaining of obstinate constipation of his bowels, general weakness, pain in the abdomen, shortness of breath, and great tympanites. He complained of having a passage of his bowels only once every few weeks. He was admitted as a patient under the care of Dr. Vischer, one of the attending surgeons of the hospital, under the supposition that there was a large abdominal tumor or some obstruction of the bowels that had led to the enormous distention of the abdomen. He was etherized, and, to the credit of Dr. Vischer, it must be said that a simple enlargement of the colon, without any other complication or tumor formation, was made out. It is evident that the case was carefully studied by the physicians of the Hahnemann Hospital, and the two accompanying reproductions of the photographs, one showing the front and the other showing the side view of the patient, were obtained through the courtesy of Dr. Vischer. These photographs convey most excellent ideas of the physical condition of the patient, especially as they were taken within a short time of his death.

The patient was dismissed from the hospital, but frequented for some time the dispensary of the same, until the day of his death, when he was found dead in the water-closet of a restaurant, as stated, with medicine in his pocket which certainly did not kill him.

Autopsy, twenty-four hours after death. Body of very peculiar configuration. While abdomen and chest were enormously distended, the body was that of a very lean subject. Bodily height, 5 feet 7½ inches; total body weight, approximately, 150 pounds. Circumference of abdomen on line of umbilicus, 7 feet 2½ inches; circumference of chest, below nipples, 5 feet and a fraction of an inch. (The photographs presented give a quite adequate appearance of the shape of the body, yet the dimensions of the thoracic and abdominal cavity were greatly augmented at the time of death, or, rather, at the autopsy.)

The skin had a glossy, pale appearance, and about the abdomen there were some post-mortem discolorations of the integument, most marked in a broad line about five inches wide, going obliquely from the right hypochondriac region toward the left groin. The abdomen was distended by gas like a drum, and a large quantity escaped on incision. Section of the integuments of the body revealed nearly complete absence of the panniculus adiposus. In fact, there appeared to be general absence of adipose tissues, not only in the trunk of the body, but also in the integuments of the extremities, being even more marked in the latter. There was also a general atrophy of the muscles of the body, which in the extremities were reduced to about one-third of their normal volume.

The peculiar disfigurement of the body was found to be due to an enormous distention of the abdominal cavity with huge increase of its viscera, encroaching upon the thoracic cavity. The spine was unduly straight. The ribs were normal in number, but anomalous as to direction of their growth. They all extended at right angles from the axis of the spine, which, as stated, was remarkably straight. In other words, the ribs—even the lower ones—pointed upward instead of downward; and the sternum, which was unusually short, measuring only 4½ inches, pointed at an angle of 90 degrees from the cervical portion of the spine.

The peritoneal sac was found to extend, having pushed up the diaphragm, as high as the third intercostal space anteriorly, and the fifth dorsal vertebra posteriorly. The thoracic cavity was reduced to about one-third of its normal capacity. The heart was normal in every respect, but occupied a perfectly horizontal position to the axis of the body, the apex occupying about the second intercostal space to the left side of the sternum.

The section of heart and aorta did not reveal anything abnormal, all the valves and orifices being perfectly consistent, and the musculature of the walls perfectly proportionate.

The blood was absolutely fluid in the cavities of the heart, as well as throughout the rest of the body, indicating that death must have been instantaneous. The pleural sacs contained no fluid, and, as well as the pericardium, showed nothing abnormal except diminution in size. The lungs, likewise, occupied a horizontal position, the apices pointing toward the spine, whereas the bases of these organs faced the sternum lying between the first and third ribs. Sections of the lungs showed them to be highly congested from passive hyperæmia and somewhat œdematous; otherwise the parenchyma of the organs was normal.

Abdomen. No excess of peritoneal fluid, although the surfaces of the peritoneum had an unusual degree of moisture. The color of the surfaces was normal; no evidence of hyperæmia or inflammatory conditions in any part. A most striking appearance was presented by the colon. It was distended by fecal contents and by gas, and although occupying a normal direction in the abdomen, it was of hugh dimensions and occupied large portions of the thoracic regions of the body. By a rough estimate, it had the appearance of being at least ten times wider than normal, the exact measurements being as follows: Total length of colon, 2.52 metres (about 8 feet 4 inches). The rest of the figures relate to the circumference of the bowels: Cæcum, 26 cm.; colon, ascending part, 37 cm.; colon, transverse part, gradually increasing from 38 cm. to 76 c.m.; colon, descending part, 60 to 62 cm.; sigmoid flexure, 62 to 69 cm.

The whole of the colon thus presented a gradual increase in size or width from the cæcum to the sigmoid flexure, the greatest increase in width being in the transverse portion.

There was no distinct angle or "M"-shaped figuration of the colon or the

so-called kinking of the bowel, as has been described in some cases. In fact, no mechanical, visible, or obvious reason for obstruction could be discovered in any part of the tube.

The colon contained two and a half pailfuls of feces, in weight about forty pounds. The physical character of the feces appeared to be normal; a semifluid, dark-brown mass with perhaps a somewhat greenish tinge; microscopically and chemically it did not present anything abnormal.

The rectum was perfectly normal in dimensions, was quite thick in its muscular coats, and presented a striking transition from extreme dilatation of the sigmoid flexure, and the rest of the colon above, to that of contraction, although no abnormal anatomical appearance of contraction that could have led to obstruction could be discovered in either the rectum or anus.

This remarkable excess in size was, however, limited to the large intestines; the small intestines, as well as the rest of the alimentary canal, being of normal dimensions. The vermiform appendix also was normal, measuring 15 cm. in length and 1 cm. in width. It was patulous, and contained a small amount of fecal concretions.

The coats or walls of this specimen of large intestine, even when examined fresh, did not appear to be unduly thick; in fact, presented to the naked eye no alterations except those referring to the muscular coat, which will be described below. There was a notable absence of sacculations throughout the whole of the colon, evidently due to an abnormality of the muscular coat, and a remarkable lack of epiploic appendices. I have stated before that there was a nearly total absence of visible adipose tissue, and this particularly holds good for the peritoneum and omentum. The omentum was ill-developed, and was found retracted or rolled up toward the diaphragm. The whole alimentary canal, with contents intact, was removed, ligatures being put at the œsophagus and rectum, and, without the liver, proved to present the actual weight of 57½ pounds, and although, unfortunately the colon was not weighed by itself, at least 47 pounds must be allowed for its weight; for the stomach and small intestines, being normal in size, hardly represented a weight exceeding 10 pounds, as will be seen below.

Stomach. Contained some undigested food, which presented the appearance indicating that death had ensued after a full meal. The stomach was of abolutely normal dimensions, and showed no pathological change except some thickening of the mucosa with mamillation or folds, and microscopical sections revealed a simple hypertrophic condition and catarrhal change of the gastric and peptic glands, and a decided increase of connective tissue in the submucosa. The muscular coats were also somewhat increased in thickness. The œsophagus was normal. The duodenum and small intestines did not present anything visibly abnormal, either as to length or caliber, and microscopical examination made from the fresh specimen showed nothing beyond some slight catarrhal change, and some increase in the volume of the tissues in both the submucosa and muscular coats.

Liver. 3½ pounds. Shape of organ somewhat deformed, the left lobe being nearly as large as the right.

Gall-bladder, with contents, normal, and ducts likewise.

There was nothing noteworthy about the liver. Its color, consistence, and surface presented nothing abnormal.

Pancreas and duct normal, there being only a few hemorrhagic foci in the glandular structures. Both liver and pancreas were located in a region normally belonging to the thoracic cavity, approximately as high as the fourth or fifth rib or intercostal space.

Spleen. 6 ounces. Normal in appearance, but in a position at least six inches higher than normal, and unusually movable and without any contact or proximity with the left kidney.

Kidneys. Normal. Supra-renal bodies normal.

The *semilunar ganglion* and a few other nerve plexuses were secured for microscopical examination, the result of which was rather negative: nothing abnormal could be seen, except, perhaps, some undue pigmentation of the ganglionic cells, a condition that is sometimes observed in perfectly normal cases.

I omitted to state that the meso-colon was abnormally large and thick—which, however, was perfectly consistent with the enormous hypertrophy of the colon.

Urinary. Bladder, ureters, and generative organs perfectly normal, except that the left testis, which was larger than the right, showed a few small cysts.

Brain showed nothing abnormal.

Cord, not examined.

A close search was made for any evidences of obstruction of the bowel of recent or old origin, but in no part of the intestinal tract was any lesion discovered that could or would have led to obstruction.

The great hypertrophy of the colon thus finds no explanation by or in any morbid or acquired anatomical lesion. Examination of the walls of the large intestine failed to reveal any morbid changes whatever, beyond a numerical hypertrophy.

I much regret that the examination was not made at a time when the specimen was fresh—on account of our anxiety to preserve the specimen unmutilated, and to enable us to make a dry preparation after the specimen was inflated to exactly the same proportions as found at autopsy. Only subsequent to this, and after the specimen was fairly dried, a microscopical examination was made of three parts of the colon—viz., ileo-

cæcal valve, transverse colon, and near the rectum. Although the examination of these specimens, which had to be re-moistened, did not yield quite satisfactory microscopical sections, yet they presented the same appearances as did a normal colon prepared under the same circumstances and conditions.

The naked-eye appearance of the specimen when fresh failed to indicate or suggest other structural change than that of bulk or size; in fact, the walls of this colon, if anything, are somewhat thinner than normal.

From a morphological standpoint it may be said, however, that besides the giant growth this colon presented an abnormality in the distribution of the longitudinal bands of muscular tissue, and possibly also of the circular coat. The well-known three bands, or tæniæ, that traverse the colon longitudinally are unusually ill-defined; in fact, they appear to be missing in certain parts of this bowel, or are so uniformly dispersed that they have lost their individuality.

The lack of the prominence and effects of these muscular bands easily explains the rather imperfect sacculation of the walls of this colon.

From the above, it is evident that we deal here with a uniform hypertrophy of the colon, which might be defined as a numerical hypertrophy without morbid change or morbid cause except those of excess in bulk or size, and we must class this specimen as one belonging in the category of giant growths or *monster per excessum*.

Literature. An examination of the literature of this subject, both as regards the congenital hypertrophy or dilatation, or constipation leading to dilatation, revealed but a few cases in which the enlargement of the colon had reached such dimensions as in the case under consideration; and, in fact, no case in all its bearings parallel to this was found.

The cases of congenital anomalies recorded refer to large diverticula of the colon. The most interesting of these was described by Grayetz, in 1876 (Virchow's *Archiv*, vol. lxviii. p. 506), in which a large diverticulum formed the contents of

a large hernial sac, the rest of the colon presenting only a moderate dilatation.

2. The case of Gruber (Virchow's *Archiv*, 1872, vol. lxii. p. 432) represents a case of an enlarged sigmoid flexure, which measured from 10 to 13 cm. in diameter, whereas the rest of the colon showed only moderate dilatation, its total length being 1.32 metres. There was displacement of the sigmoid flexure to the upper part of the abdomen and very marked chronic peritonitis, adhesive bands having been instrumental in dragging the flexure from its normal position.

3. Little and Calloway (*Transactions of the Pathological Society of London*, 1850, vol. iii. p. 107) describe the case of an imbecile, male, aged thirty-four years, 6 feet in height, whose abdominal measure in the region of the umbilicus was 45 inches around, and who was admitted to the hospital on account of long-standing obstipation.

He died seven days after admission. The autopsy showed the transverse colon to be 6 inches in diameter (about 19 in circumference), while the descending and sigmoid portion measured 12 by 20 inches in diameter. There was decided colitis and thickening of the mucous membrane, and numerous ulcerations.

4. Peacock (*Transactions of the Pathological Society of London*, vol. xxiii. p. 105) records the case of a male, aged twenty-eight years, teacher of vocal music, who had suffered from constipation since birth.

He frequently resorted to enemata, without which there would be no evacuation of feces but much flatus, which necessitated his frequently leaving the room to expel wind. He enjoyed good health up to six weeks before death. He was said to have died from heart-failure.

The autopsy showed that every organ in the body was normal except the colon, which was enormously and uniformly distended, measuring 6 to 8 inches in diameter, and containing fifteen quarts of fecal matter, semifluid in consistency and green in color. There was also decided chronic colitis, with extensive ulceration. The rectum was not involved.

5. Levitt (*Chicago Medical Journal*, 1867, vol. xxiii. p. 359) gives an account of an interesting case:

A white male, aged twenty-one years, suffered from obstinate constipation that would last eight to ten days at a time for nine years. When he came under medical observation he suffered great agony from pain in the abdomen, with frequent desire to expel flatus, which could only be accomplished by standing on his head and hands to give his body a perpendicular position.

The abdomen was greatly distended and tympanitic. Death resulted from peritonitis. The peritoneal cavity contained fecal matter, due to perforation of the colon in several places. The ascending and descending portions of the colon lay as large columns, side by side, for there was no evidence of a transverse part of the colon. The colon was filled with soft fecal matter, measuring about a pailful. The mucous membrane of the colon was much thickened and ulcerated, which had led to perforation and peritonitis, as stated.

The measurements of the colon are given as $5\frac{1}{2}$ inches in diameter in both its ascending and descending parts. The accumulation of feces appears to have been most marked in the sigmoid flexure, which pressed upon the rectum with such force as to prevent defecation, and by this the author of the paper explains the peculiar position which the patient had to assume to expel flatus or feces.

6. Cruvelhier (referred to by Lichtenstein in Ziemssen's *Cyclopædia of the Practice of Medicine*, vol. vii. p. 609) speaks of a case of an old woman suffering from coprostasis, in which the colon was distended to the size of a horse's colon and was filled with thick feces.

The same author refers to the case of an old man affected similarly, in whom the colon measured 35 cm. in circumference, the cæcum being the size of a child's head.

There are numerous other cases in literature which refer to coprostasis of long duration, lasting as long as seven months; of individuals having a passage of feces only a few times a year, with more or less dilatation of the colon, but in most of which

there appears to have been some ulceration of the mucous membrane. But a description of the cases is so deficient, or in some instances so inaccessible, that no use can be made of them. Moreover, they have no direct bearing upon or similarity with my case.

There are, also, some doubtful cases in the older literature in which indefinite coprostasis is spoken of, and in which enormous quantities of feces, weighing as much as sixty pounds, are referred to.

Lichtenstein (Ziemssen's *Cyclopædia of Medicine*, 1876, vol. viii. p. 583, American publication) speaks of coprostasis conditioned upon habitual constipation, which he terms *ileus paralyticus* in the small intestines, parallel with coprostasis of the large. He attributes this condition solely to insufficiency of the forces destined to move the contents of the intestines forward, want of peristalsis, and ultimate paralysis of the bowel, which leads to arrest of the passage of feces. This, with insufficiency of abdominal pressure, leads, in turn, to dilatation of the bowel.

Undue sacculation of the colon, due to contraction of the longitudinal muscular coat or the failure of the proper development of these muscular bands, and likewise an abnormal mesocolon, are considered important factors of coprostasis and subsequent dilatation of colon.

I am indebted to Dr. R. P. Harris for the following note, which has some interest in illustration of the cases on record, but does not correspond in all its features with the case under consideration.

"*Fatal constipation from neglect* is most apt to occur in lunatics living at home, where their habits are not closely looked into, and the measure of their discharges is not known by those who reside with them. One such case came under my observation in 1848, in a single lady of thirty-eight years of age, who had been the subject of melancholia for twelve years. She had been in the habit of visiting a yard-privy by herself, and it was not known to her parents that she did not evacuate any fecal matter. When taken sick to bed she had frequent peristaltic

pains, like the early contractions of labor; passed small quantities of softened fecal matter; was affected with hæmorrhoids, and her urine dribbled from her. From the caput coli to the anus her large intestine was packed with solid fecal matter which could not be removed, and she died in five days after I was called in.

"An autopsy showed that her rectum was so distended that it filled the pelvic cavity, pressed the uterus against the pubic bones, the organ being white and bloodless, and prevented the filling of the bladder. The fecal matter in the rectum was as hard as brick-clay is when ready for the mould, and the whole of the large intestine was distended to a diameter varying from 3 to 4 inches.

"She must have been constipated for a number of weeks."

Résumé.—A congenitally large tube, like the colon in this case, with anomalous arrangement of the muscular tissue, is naturally unfit for contraction or any necessary physiological exercise, probably for want of proper innervation; in other words, the colon presenting a large cavity whose walls were not under physiological control, it is perfectly natural that the regular expulsion of feces would be abnormal.

It is natural to suppose that, under such circumstances, it would take a great accumulation of feces to fill the cavity, and that such accumulation would take an abnormal time to be expelled. Many days might elapse before the accumulation would be sufficient in bulk to make an impression that would promote a physiological contraction sufficient to expel it. It is thus plausible that an evacuation of the bowels might be deferred for weeks, or even a month, as in the case under consideration.

It is evident, from the study of this case, that we deal here primarily with a congenital anomaly, due to an anomalous arrangement of the muscular fibres of the longitudinal as well as circular coats of the colon. Contrary to the usual rule, where constipation or coprostasis would cause a dilatation of the walls of the gut, a dilatation of the gut congenitally malformed and distended was the factor in causing the coprostasis or the accumulation of feces in this case. The absence

of sacculations and folds which form the physiological cause for the accumulation and hardening of feces was also wanting in this case, and thus the normal stimulus upon the mucous membrane of the colon was missing.

Whereas, there was no evident histological evidence of any change in the nervous structures in this case, it appears to me probable that the want of proper innervation of the colon—in other words, a congenital paralysis of this bowel—was a very important factor in producing the dilatation as described.

I wish to acknowledge the aid, in the study of this case, of my assistant, Dr. J. L. Wethered, who aided me at the rather voluminous autopsy and the subsequent study of the case—a work which consumed a great deal of time.

THE BLENDED TOCCI BROTHERS, OF LOCANA, ITALY.

By ROBERT P. HARRIS, M.D.

[Read May 4, 1892.]

Dr. Harris brought to the notice of the College an enlarged photograph of this remarkable monstrosity, which had been prepared for the Mütter Museum at the desire of its curator, and made the following remarks:

We have here a nude representation of what we must regard as the most remarkable duplex monstrosity that the world has seen since the death, three hundred and seventy-four years ago, of their Scotch analogue who had reached the age of twenty-eight years. This peculiar type of blended twins appears, on an average, twice in a century, as there have been about a dozen in the last six hundred years, and but two in the current century. The most remarkable feature about the Locana twins is that they are living and in good health at the age of fourteen and a half years, and bid fair to reach mature age, because of the perfection and independence of their thoracic and abdominal viscera. In the last six hundred years but two monstrosities of the same type have lived out their first year; and this early mortality we must attribute to a want of internal anatomical symmetry, and particularly to an abnormal construction of the heart and distribution of the bloodvessels in one twin. It is doubtful if united twins are ever equals in mental and physical vigor, and the Tocci Brothers are as nearly alike in health and strength as has been the case in the subjects that have lived the longest.

The photograph before us represents the boys in a standing

position, but it will be seen at once that they are mainly sustained in it by the use of their arms, and that this is more markedly the case with the left twin, whose shoulder is forced upward, because of the weak support given by his club-foot and imperfectly developed leg.

These twins were born in Piedmontese Italy, on October 4, 1877, after a labor of eight hours, under a midwife, the head of the right boy, Giovanni, coming first; and he appears to have held that relative position, in a mental sense, ever since. Giacomo's head soon followed, and then came the double thorax, a single abdomen, one pair of legs, and a single placenta. Nothing was said about the cord, except that there was but one; but it was no doubt composed of six vessels—four arteries and two veins. The twins weighed $8\frac{3}{4}$ pounds when a month old, and probably a pound less at birth. When three years old, as shown by a photograph, they had narrow shoulders, a corpulent abdomen, and, for their age, large testicles. Giovanni had a long face and a girl-like appearance, but his head-circumference has always been a little the larger of the two.

These xiphodidymi belong to the class that is distinguished by having two heads, four arms, and only two legs. In general outline they resemble, when their legs are together, a letter Y—the heads, shoulders, and chests down to the sixth ribs making the V, and the abdomen and legs the I, or stem. Their present weight is 95 pounds, which is about that of a healthy, robust boy of their age. Their arms, having much more exercise than their legs, are larger, in proportion to their age, than the latter; and the glutei muscles of Giacomo are badly developed, because of his talipes equino-varus, and consequent inability to develop them by exercise. Single boys of fourteen, as a rule, have much better developed legs than arms, which led to the expression in war times: "Better fitted for running away than handling a musket." But the Tocci boys are the reverse of this in strength.

If we had a back view of the monstrosity, it would show their two inner arms crossing each other over to their outer

shoulders, in the position in which they usually hold them; two converging spinal sulci extending down to two sacra; two outer nates, as in a single subject; and two little rudimentary nates with a cleft between them, located over the intra-sacral symphysis. They have no rudimentary nodule to represent an attempt at the formation of a third leg, as has been found in some analogues.

As they look at you, the two boys are quite different in facial contour, and Giovanni is generally credited with having the better mind; but their faces in profile bear a closer resemblance. They have fair skins, with at times a rosy color, and thick, brown hair. Their bodies are short, and they are below the medium height for their years. Their facial expression is not a happy one, when in repose, and reminded me of what I have noticed in boys having deformed feet—a shame-faced look.

In measure of health the twins compare well with normal children of their own sex, having had but little sickness since birth, and not having lost a day therefrom since they commenced to exhibit themselves in the United States, six months ago.

Giovanni is the stronger and more erect of the two, has the better ear for music, learns a foreign language the more readily, and is generally the more intelligent of the two. He has a natural talent for drawing, and is devoted to making pictures of our domestic animals, such as the horse, cow, etc., and of some of the savage quadrupeds, as of the lion and tiger. Giacomo is the critic in art, although drawing but little, and his taste is for caricatures. The brothers converse a great deal together. They are both right-handed, although one might have supposed that Giacomo would have naturally preferred his free arm, the left.

The boys have each two lungs, the outer being the larger, and are forced to breathe largely by their diaphragms. They have separate and distinct hearts, located in the left chest-cavity respectively, and these hearts are believed to be normal in structure. Giovanni feels his heart beating on the left side—and if either boy had an abnormal cardiac structure, he should be the one—yet his color and health indicate the contrary.

The cardiac beats are not synchronous, and one heart generally pulsates a little quicker than the other.

They have two stomachs, and that of Giovanni is said to be reversed, the greater curvature being to the right, as was the case in Rita, of the Sassari girls of 1829. I was not permitted to verify this, because of the opposition of the father. These two stomachs are as independent functionally as if they occupied two different abdomens. Recently, when travelling by railroad, one of the boys became very pale, and directly vomited the contents of his stomach,[1] while the other was so entirely free from nausea that he laughed at his brother for his mishap. One brother may wake up hungry, drink a cup of coffee, and eat something, while the other remains asleep. The two stomachs do not appear to be influenced in the least by being in contact, but only by their respective pneumogastric nerves.

There are evidently two sets of intestines—large and small. One boy can have a desire to defecate when the other has not; and this is particularly the case when one has a diarrhœa, in which event he only has a discharge, while the other is passive. There must therefore be two colons, as were found on autopsy in the Padua boys of 1691. They have probably a common rectum, as had also the Padua analogue; but it is possible that this part of the bowel may be bifid, which would be an interesting feature to determine by touch or speculum. The twins, from habit and convenience, defecate almost always at the same time.

They have two bladders and one urethra, as had also the Padua boys, although they usually urinate together. As their tastes for food and desire for drink are not the same, one boy may be awakened from his sleep in the morning by a distended bladder and empty it without waking up the other, in whom the kidneys have been less active.

My catechetical examinations made last month confirm the opinions respecting the anatomy of the twins that were formed after auscultation, percussion, and a knowledge of their habits,

[1] This was repeated by Giacomo in public on April 23, 1892, at the Dime Museum, Philadelphia.

by Drs. Fubini and Mosso, of Turin, in their second month, and by Drs. Colrat and Rebatel, of Lyons, in their thirteenth. The conjectural belief that they might have two colons has been changed into one of knowledge, as shown by their independence in defecation. In the event of an autopsy, these colons will no doubt be found of small calibre, and quite abnormal as to length and direction. We are warranted in this belief by discoveries that have already been made in the examination of dead analogues. Even the single colon of a double monster is quite abnormal.

The boys are quite differently affected by changes of temperature. Giovanni requires less underwear than his brother, and will perspire freely on a hot day, while Giacomo has a dry skin. Either brother may be seized with an attack of coryza, as the effect of a direct wind-draught, when the other entirely escapes. They sleep upon the back—or, more correctly, each is in a dorso-lateral position, and places the side or back of his head upon the pillow. They usually sleep eight or nine hours continuously. For a change of position they sometimes turn over upon their abdomen for a short time, but never sleep in this form of decubitus.

A prick with a pin in the median line of union is felt by both brothers, but the sensation is lost to one twin in passing to either side. The penis is said to have a sensation common to each, and the scrotum has a partial one beyond the median line. It is claimed that one twin feels a little when his brother's testicle is touched, which I believe to be an error, the sense being in the skin only, as the testicles are supplied with nerves through the inguinal canal. The penis and testicles are in an undeveloped state, being small for their age. The penis becomes erect, but the boys have no knowledge of its sexual function.

The two legs are entirely independent, and each belongs to, and is controlled by, the boy whose head is on the same side. One boy does not feel a touch upon the other boy's leg, and has no power, by his will, to give it the least motion. It is possible that the twins might balance themselves so as to stand, as their Scotch analogue is said to have done, if the leg and

foot of Giacomo were as well formed as those of Giovanni, and had the same degree of strength that the latter appear to have.

Whether the boys have two separate livers, or a double one with two gall-bladders, has not yet been ascertained, for want of permission to make the required examination. Having two bladders, they may have two pairs of vesiculæ seminales, but are more likely to have one to each bladder, with one vas deferens and one ejaculatory duct, as this would be in correspondence with the existence of one testicle to the owner of each bladder, and one urethra for the exit of the ducts. I find no reference to the seminal vesicles in any reports of autopsies made in male analogues.

With regard to the other viscera, we can only form an inferential opinion, as follows: The spleens are small, and located right and left to correspond with the positions of the stomachs. The pancreases have their heads facing each other, to correspond with the curve of each duodenum. The kidneys are in two pairs, the outer being large and the inner being small, or, perhaps, rudimentary. It is rare to find no trace of the two inner kidneys where the spinal columns are well separated so as to give space for them.

I see no reason why these Tocci boys may not live a number of years yet. Giovanni is the stronger, mentally and physically, but the difference is not much marked, and no special element of weakness likely to shorten life appears to have been discovered in Giacomo. They have both learned a certain measure of French and German, and can both sing, Giovanni having the higher-pitched voice. One of the Scotch twins already mentioned as having reached the age of twenty-eight, is recorded as having been quite stupid when compared to his brother. We have no such difference to record here.

NOTES ON IRRITABLE HEART IN NEURASTHENIC CASES, AND THE EFFECT OF LIMITED MUSCULAR ACTION ON THE HEART IN HEALTH AND DISEASE.

By JOHN K. MITCHELL.

[Read May 4, 1892.]

Many patients suffering from "general nervousness," or "neurasthenia," will tell the physician, who makes a physical examination of them, that they are annoyed by palpitation or by shortness of breath on very trifling exertion. Probably the most careful study of the heart's sounds or action will reveal nothing. With the patient lying down it beats steadily, it may be a little too fast; the arterial tension does not seem to vary from the standard of health, there is no accentuation of either sound, and no trace of murmurs.

But if the patient makes a movement, even while still supine, the frequency of the pulse increases at once, the first sound becomes a trifle shorter, the second gets a slight but distinct accentuation, and for a space longer or shorter, according to the effort made, these symptoms continue. Usually, if the exertion has been slight, the heart quiets again after ten or a dozen beats.

After I first noticed this I was accustomed to ask the patient, lying perfectly still, to make some such small sharp effort as closing the hand tightly once. If the heart be at all unusually excitable, this will produce a marked effect on its sounds and action, and I find it a useful, if not a very important aid, to the diagnosis. Sometimes a murmur will be produced not otherwise audible. One might find the same murmur if the patient

were to rise suddenly from a recumbent position, but if, as is commonly found, it lasts only during five or six beats, we should scarcely find time to apply the stethoscope after the change of position, before it would have disappeared. It must be some very slight motion which is made, because in a man with a not excessively sensitive heart to raise the arm above the head will hurry the beat decidedly.

The first case in which I took especial notice of this condition is reported as follows in my note-book, omitting the general history, which was one of the innumerable cases of break-down from overwork. There was no history of excess in the use of alcohol or tobacco.

Case I.—W. J. D. He was easily tired, somewhat morbid about himself; suffering from a mild degree of insomnia, not anæmic, and had palpitation of the heart upon every slight exertion. Standing, his heart beat 72; lying, 60 per minute. After he had lain motionless for a few minutes, and the pulse was steady at 60, I examined the heart very minutely. Both sounds were less strong, less well outlined, if I may use the phrase, than they should have been, but there was no trace of murmur. On sitting up, the force and frequency increased for eight or ten beats, and a soft but distinct blowing sound was heard at the apex, beginning just after the first sound and ending with it. After the first few seconds it was inaudible, but could be brought out clearly for five or six beats by raising the hands a little.

Erect, although both the force and frequency were again increased, the murmur was not audible, nor could it be reproduced when in this position, even by a good deal of muscular effort.

When lying down, the heart was very little affected by slight movements of the hands or arms, nor could the murmur be detected.

This case, a less typical one of the irritable state of which I am speaking than the others reported below, set me to looking for and studying the peculiar condition, and from a considerable collection of notes I append four other examinations.

Case II.—A. C. C. A hard-worked and worried bank officer. Never very strong, but had had no illness until a break-down one year before examination.

Examination. Cardiac outlines normal; apex-beat faintly tangible in fifth interspace; both sounds feeble, the first especially lacking in sharpness, and the second shorter than normal. Pulse, recumbent, 80; regular, but decidedly weak, with ill-filled arteries. The slightest motion of the hand, as extension or pronation, accentuated both sounds very markedly, the first even more than the second, and increased the beat to about 90. Even then the

arteries were less filled than they should have been, and the action of the heart, to the touch, not much stronger. Erect, the force was greatly increased for a few beats, and the rate first exceeded and then remained at 110 a minute.

Case III.—C. H. K., aged thirty-three years. Unmarried woman, physician. Worry and overwork had resulted in depression, with suicidal tendencies, and constant severe headache. There were no symptoms referred to the heart. *Recumbent*, the pulse was slow, full, regular, and 62 a minute. Making a fist once, caused a rise to a rate of 74, and the experiment several times repeated gave similar results. There appeared to be no increase of accentuation. The further exertion of raising the arm increased the speed only two or three beats more in a minute. *Erect*, the beat was 66 to 68.

Case IV.—W. J. R., single, male, aged twenty-three years, student. Break-down at school, repeated in college, and constant overwork at high pressure had resulted in extreme general nervousness, with physical prostration and extraordinary insomnia. There was no anæmia found on examination of the blood.

Examination. There was no increase in the size of the heart; apex in the usual situation; impulse very forcible and thumping. *Recumbent*, there was still over-action, rate 112 per minute, with a tendency to split the first sound. To make a fist increased both force and frequency, and after the effort there was always an interval of six or eight beats before any effect on the action. Then the beats became more rapid for ten to twelve beats, and quickly dropped again to the former speed. Counted for half a minute succeeding the motion of the hand, the rate rose five or six beats in that period. In the next half-minute it fell again to the same number as before the movement.

Sitting, after the first exertion of coming to the sitting position passed—the heart ran at 120. Occasionally, for three or four beats together, a very faint whiff, beginning just after and ending with the systolic sound, audible only in the area immediately about the apex and not traceable in any direction, was heard, occurring irregularly at intervals of from 25 to 40 beats.

Standing, this systolic murmur was a little better heard in the mitral region, but could not be followed elsewhere. The speed increased to 140 a minute while erect.

After noticing this murmur I made the patient lie down again, and found that although when he was perfectly still the systolic whiff was inaudible, it could be reproduced at will by making a fist once or twice. To raise the arm above the head brought it out clearly, too.

Professor DaCosta saw this case in consultation with me, and detected the same murmur, though, as the patient had been at rest in bed for nearly two weeks, it was less frequent and the heart somewhat slower than when first examined. Dr. DaCosta thought as I did, that the murmur was clearly functional in its nature, although the situation was not that of the murmurs

usually described as "anæmic," between which and "functional" murmurs a distinction should be made.

The patient had had, and at this examination, six weeks after the first inspection, still had, a curious symptom: a general surface flush, most evident in the trunk, without rise of temperature and with no subjective sense of heat. It appeared at irregular intervals during the day, and disappeared slowly. It was easily dissipated by deep massage or by effleurage. His pulse, even when considerably excited, was habitually reduced by the use of general massage from ten to eighteen beats a minute. This seems to be usually the case where a rapid heart action is part of a general nervousness, as I have observed the same result in several other persons in whom a like condition of cardiac irritability and over-action was present, and in some cases of nervous palpitation it is found that the heart is steadied by exercise. The flush was no doubt an indication of what may be called arterial incompetence—a weakness or lack of tone in the vessel walls.

Case V.—J. H. M., aged thirty-three years. Business man, formerly active, fond of exercise, interested in his work; now irritable, hypochondriac, disinclined to physical exertion, and generally nervous. The patient says his heart is often intermittent, but no such irregularity was observed.

Examination. Recumbent, no over-action, no murmur, and the sounds in no way abnormal. Making a fist changed the character of the sounds, rendering them sharper and more valvular at once, but produced no murmur. Previous to the movement the beat was 88, in the succeeding minute it rose to 100. After repeating the effort two or three times with the same effect the irritability disappeared or was exhausted, and the motion brought about no change in the rate or sound, and to raise the arm did not make any evident change in the rate or action.

This observation raised in my mind a question which I could nowhere find satisfactorily answered, as to the cause of the increased rapidity of the cardiac action, and the distinct change in the sounds following so slight an exertion.

At first I thought the force of the beat, as well as the speed, was greater, and although this was not borne out by the subsequent observations and tracing of the pulse, it may still be that the force of the *heart* is increased, while the arterial pressure is unchanged or lessened.

The alteration in the character of the sounds is to the ear very decided. The confused dull first sound is made more defined and sharp, the second sound gains an accentuation, and, as noted in some of the cases, a murmur, otherwise only occasionally and indistinctly present, is clearly brought out.

The action which the patients made was, as I have said, a single sharp closure of the hand opposite that on which the sphygmograph was, lasting a couple of seconds. Could so slight a muscular action affect the blood-supply of the heart enough to cause the changes observed? and how?

Guy was one of the first to show that the increase of the heart rapidity in the erect over the recumbent posture was due solely to muscular action; this he demonstrated by bringing a man from a horizontal to a perpendicular position without movement on the part of the subject. In a person in ordinary health, the difference in pulse rate between lying supine and standing, is about twenty beats a minute. In the most excitable hearts among the cases here reported, this difference amounted to thirty beats. (Cases II., IV., and V.)

In cases II., III., and V., the increase brought about by making a fist was ten to twelve beats per minute. Yet in two of these persons, to stand upright increased the speed only about thirty a minute, and in the third the difference between the pulse recumbent and erect was but eight beats in the minute, so that the trifling exertion of clenching the fist had a greater effect than the effort of rising and continuing to stand[1]—a curious fact for which I am as yet unable to account.

I framed for myself several theories to explain these strange results, with very little satisfaction. My first supposition was that the slight effort made threw more blood upon the heart, calling for increased exertion on the part of that organ—but when the physiology of the circulation was considered, this was presently seen to be improbable; first, because the amount of blood sent to the heart by so trifling an exertion as supination of the hand (Case II.) must be almost incalculably small; second, the tracings of the pulse show a slight but evident decrease of pressure to follow the "making a fist." Whether this decrease be only arterial, or cardiac as well, is yet to be determined. Exactly the reverse may be the true cause, namely—and Dr. Newell Martin suggested this to

[1] In health, the difference between lying and sitting is about five beats, and between lying and standing (as mentioned above) about twenty beats per minute.

me in conversation as a theory which might be considered—that the effort made decreases the blood-supply in the heart, and increased rapidity results from the stimulus caused by this deficiency.

The tracings bear out the clinical observation that a considerable interval lies between the effort made and the resulting decrease of pressure and increase of rapidity. This is longer when very slight movement is made; shorter when the movement is a large or more strenuous one. For instance, in tracings from my own pulse, which is regular, with a medium tension, and beats, when lying down, about 72; there is an interval of nearly six seconds in one of the tracings and of five seconds in the other; that is, counting by beats, eight beats in the former and six in the latter case. The action was the same in both. When the whole arm was raised the interval before the change appeared in the pulse was each time almost exactly three seconds, or four beats, and about the same time elapsed when a slightly greater muscular effort was made, such as to extend and hold extended the leg. After the cessation of effort the return of the pulse to its normal is gradual, and takes place in from eight to fifteen beats. Continued contraction in this way of a single group of muscles has no greater effect on the blood-pressure and rate, and the tracing becomes gradually the same as before the exertion, even while the effort is continued.

The time-interval between effort and effect presents a question of some nicety for settlement. There is no study known to me of the time required for a peripheral stimulus to reach and affect the heart, whether that stimulus be vascular or nervous. Even the rapidity of the return of the blood from the extremity of a limb to the heart is a matter on which physiologists are not agreed, nor is it so easy to measure as the period between an excitation and its effect on the knee-jerk, for instance, and I have therefore not laid any stress on this point. It should be said that during the making of these tracings, we had not at our disposal any means of automatically registering upon the same paper the moment when the movement was begun.

Hence the time indicated is that when the order was given. There are, therefore, *two* intervals, the sum of which make the interval on the tracing—one the reaction-time for the nerves of the part moved, one the interval between this motion and the cardiac effect.

Several papers in the series of studies from Marey's laboratory[1] bear on these points, and remain to be discussed later.

When the pulse of the patient was very fast, small efforts produced a less effect, as in case IV., where repeated examinations showed that the increase in speed was only six beats a minute from making a fist, a change about the same as occurs in a healthy heart from the same exertion. In all the cases the increase was only to be observed when the subject was recumbent.

Sometimes with a tumultuous, over-acting heart, a slight effort seems even to decrease the speed and steady the action, so that a murmur previously ill-heard from the confusion and irregularity of the sounds, is clearly brought out. In the rapid irregular heart so common in choreic children this has often been noticed at the Infirmary for Nervous Diseases, and it is a matter of common observation that in some cases of palpitation in neurasthenics, exercise causes the palpitation and oppression to disappear.

In view of these observations, it seems probable, though, without the further study which I hope will prove it, it cannot be positively asserted, that the mechanism of these changes from slight effort which I have tried to describe, is of the nature of a reinforcement by voluntary act, like that observed by Jendrássik in his study of the knee-jerk; it is, that is to say, a purely nervous phenomenon. The reasons for this view are as follows: the time-interval, which is composed of *two* factors, as before stated—the reaction-time between the order given and the movement made, and the time between the movement and its reflex (?) effect on the heart. The course of the phenomena resulting is like that of all reflex acts, an

[1] Physiologie Experimentale, Paris, 1876.

interval of lost time, a period of excitation, and a period of decline.

The excitability of the heart to these slight stimuli is soon exhausted, long before any muscular fatigue from effort could make any difference—just as other reflex actions and their reinforcements, if repeatedly produced, are presently lessened. There are other points of likeness to these phenomena; a very slight voluntary effort will cause them, as to turn the hand over. Could we measure the effect on the pulse with the same ease and accuracy as the knee-jerk, I am convinced we should find as trifling stimuli would affect the former as do influence the latter. The reinforcibility (if the use of this word is not begging the question) is present in health and exaggerated in certain conditions of disease, as is the excitability of the knee-reflex; it resembles it, too, in that emotion makes the excitability greater at first and then lessens it, as Lombard showed in the case of the knee-jerk.

An attempt was made to simplify the problem by eliminating the possible influence of the increase or decrease of blood in the heart caused by slight effort. It would seem that if the circulation were cut off, and motion still produced a change in rate and pressure, it could only be a nervous effect. Accordingly, the sphygmograph was adjusted on the right arm, the blood was removed from the left arm by a rubber bandage, and an Esmarch band tied tightly above the elbow. When no pulse was perceptible the effort was made as before. The result of the few experiments was conclusive. A marked drop in pressure took place, and a slight change in rate. The effect seemed to follow more promptly the motion made than usual, and was even more decided.

I do not wish to appear to lay too much weight on what is, after all, a small clinical point. It is easy to see, however, how useful it may prove to be to have at our disposal a means whereby we may change the action of the heart and bring out doubtful murmurs, with the patient lying in bed.

This study is as yet incomplete, but seemed in some respects of sufficient novelty and interest to warrant my bringing it

before you, and discussing it as a gross phenomenon, and chiefly from the clinical side. The nice questions of physiology involved are in course of examination, and before long it is hoped to lay before the College somewhat more certain results.

Note.—A word as to the apparatus used may not be out of place. After several efforts with the cardiograph, with the lever-tambour, and various forms of sphygmograph, in which I had the kind assistance of Professor Reichert, we finally settled upon the use of a Marey sphygmograph, writing upon a kymograph. The arm was fixed in a trough, and the subject, sometimes Professor Reichert, sometimes myself, laid so that he could not see the tracing. One disadvantage of this apparatus is that the tracing is necessarily reversed, but it proved the readiest way to secure the long tracings which we wanted.

DISCUSSION.

Dr. J. M. Da Costa: I have been much interested in this valuable paper. I had the pleasure of seeing one of the cases with Dr. Mitchell. There are one or two points to which I wish to call attention and on which I should like full information: First, were all these murmurs apex murmurs? Secondly, I would urge Dr. Mitchell to go on with these observations with the object of ascertaining whether or not the force is markedly increased or decreased. I understood him to say that it was not uniformly affected. If it were shown that the force was increased, we should have a therapeutic means whereby patients with flabby and dilated hearts could in their own rooms exercise and strengthen the heart muscle; if decreased, the exercise would be valuable in cases of strongly acting hearts undergoing hypertrophy. The site of the murmur that is developed seems to me of importance in showing that the abnormal sound is a functional murmur due to dynamic causes connected with the perverted cardiac action, and not an anæmic murmur, which is so much more generally basic. The murmur heard in the observations presented to-night is akin to that we find from excitement, as during application for life insurance, or after active exercise, such as running or rowing.

Dr. H. C. Wood: I have often found that these neurasthenic murmurs persist without distinct anæmia for months. I have seen them continue for

so long a time that I have felt forced to abandon the original diagnosis that the murmur was functional, and yet have seen after a year the murmur entirely disappear. I should like some of those who are more familiar with the physiology of the heart to give an explanation of these murmurs.

Dr. F. P. Henry: The cardiac symptoms described by Dr. Mitchell in his interesting paper are, like so many other morbid conditions, exaggerations of normal processes. The same degree of cardiac action which, in the healthy, is called forth by some decided muscular exertion, may, in the neurasthenic, be evoked by trivial causes. This is, I believe, in part due to the fact that in the neurasthenic there is a want of correlation between the action of the heart and that of the arterial walls. In health, on sudden exertion, the heart's action is accelerated; but even if the exertion be continued, the cardiac beats soon diminish in frequency, because the arterial walls relax and permit a freer passage of blood. In the neurasthenic, on physical exertion or mental emotion, the arterial walls frequently remain contracted for an abnormally long period, and often give rise to well-marked steno-cardiac paroxysms, resembling those of angina pectoris. In fact, the symptoms described by Dr. Mitchell may be regarded as those of rudimentary neurotic angina.

This view of the pathogeny of these cases is corroborated by the presence in them of transient mitral systolic murmurs, which can only be due to temporary insufficiency of the mitral valve from increased arterial tension. No other explanation of these murmurs is possible.

Many neurasthenics presenting these cardio-vascular symptoms are by no means anæmic, although at the present day one is scarcely justified in pronouncing anæmia absent until the blood corpuscles have been found, by proper examination, to be normal both as to quantity and quality.

Dr. James B. Walker: It has seemed to me, in studying weak hearts, that varying degrees of intra-ventricular pressure and varying tension would account for these transient murmurs. I had some time ago a patient who was under treatment for sarcoma of the nose. I received a note from the surgeon that the man had mitral disease. I replied that I had examined the heart frequently, and that there had been no evidence of such disease, and suggested that probably the fatigue of the operation, with the hemorrhage, had led to dilatation of the ventricle, with the occurrence of the murmur. The correctness of this view was proven by an examination made by Dr. Pepper, the operator, and myself after one of the operative procedures. I have seen many other such cases.

These murmurs which occur transiently are due, I think, to varying degrees of dilatation of the ventricle. The chordæ tendineæ are of definite length, while the distention of the ventricles may vary much. Given a weak heart which readily dilates, any obstruction or interference to the entrance of blood into the arterial system (and even so slight an exertion as that to which Dr. Mitchell has called attention may be sufficient to increase the resistance to

the entrance of the blood) may cause temporary dilatation of the ventricle, and this temporary dilatation, acting on the chordæ tendineæ, may prevent complete closure of the mitral orifice early in the systole. If while they are taut and the cavity is of normal size they just permit closure of the valve, any increase in the size of the cavity may cause a murmur, particularly at the beginning of the systole. I am satisfied that such murmurs as have been described are explainable in this way.

The President: Dr. Mitchell's observations are certainly interesting and, to some extent, novel. Dr. Mitchell appears to have made clear that a very slight muscular act may quicken the heart, lessen its force, and lower arterial pressures. His experiments seem to prove that the influence which affects the heart arises with the volitional act, and has analogies to some of the reinforcement phenomena which increase or lessen the knee-jerk.

Dr. Mitchell: In answer to Dr. Da Costa I would say that all the murmurs noted as brought out by motion were at the apex, and not basic murmurs.

Although the auscultatory observation was that the force of the heart was increased by the closing of the hand, the tracings of the pulse show uniformly a decrease of arterial pressure. The two facts, however, are not incompatible.

In none of the cases reported was anæmia a conspicuous feature. Most of them were neurasthenic patients with some physical prostration—not in good health, but not in conspicuously bad health. In several I examined the blood, and in two cases more than once, but found no marked anæmia. I did not mention these facts, because I wished to make the histories as brief as possible, and I have therefore only stated the salient points in each case.

MEMOIR OF DR. LEWIS RODMAN.

By WILLIAM G. PORTER, M.D.,

SENIOR SURGEON TO THE PHILADELPHIA HOSPITAL; SURGEON TO THE PRESBYTERIAN HOSPITAL.

[Read May 4, 1892.]

Dr. Lewis Rodman, the subject of this memoir, was born on his father's farm at Eddington, Bensalem Township, Bucks County, Pennsylvania, June 12, 1806. His father was Gilbert Rodman, who was a lineal descendant in the fifth generation of John Rodman, of Barbadoes, who was the founder of the Rodman family in America. John Rodman, 1st, in the year 1655, for wearing his hat on in the Assizes in New Ross, County Wexford, Ireland, was committed to jail by Judge Louder, kept a prisoner three months, and then banished that country. His banishment, no doubt, followed because he conscientiously refused to purge himself of the contempt of court. Immediately upon his banishment it is probable that John Rodman went to Barbadoes. He died there in 1686. Thomas Rodman, the son of John Rodman, came to Newport, Rhode Island, in 1675. He was a prominent member of the Society of Friends, and clerk of the monthly, quarterly, and yearly meetings of Rhode Island for thirty years. He was also the first clerk of the New England yearly meeting, which position he held until 1718. He was an eminent physician and surgeon, chiefly celebrated as an obstetrician, and was sent for in difficult cases to go great distances. Dr. John Rodman, the younger brother of Thomas, followed him to Newport in 1682. Within a few years after their arrival at Newport, the Rodmans, among others, directed their attention toward New Jersey and Pennsylvania, and the two brothers, Drs.

Thomas and John, made investments in land there. Penn's charter, which had just been granted, had attracted attention to that locality. In April, 1686, Dr. John Rodman purchased one thousand acres in Burlington County, N. J.; and on September 24, 1686, Dr. Thomas Rodman, his brother, purchased from James Marten a "propriety," some of it in Burlington County, some in Hunterdon County, and some in Gloucester County, New Jersey. He exchanged five hundred acres of it, July 5, 1710, for the plantation in Barbadoes, which his father, John Rodman, had devised to his daughter, Catharine Brandreth. The first of the Rodmans who owned land in Bucks County was Dr. John, 2d, grandson of the original John, of Barbadoes. He settled in Burlington, New Jersey, and practised medicine until his death, in 1756. He purchased land on the Neshaminy, in Bensalem Township, in 1712; a dwelling was erected on this in 1715, and here his son William subsequently settled and resided until his death, in 1794. The plantation was first called Rodmanda, but the name was subsequently changed to Flushing, after his birthplace on Long Island. This was one of the most notable homesteads in Bucks County, and the old dwelling, which had weathered the storms of one hundred and fifty years, was torn down in 1861 to make room for a more modern structure. One hundred and sixty-eight years ago, William Rodman stuck his buttonwood riding switch into the ground by the side of a fine spring of water near the house, and in all these years it has grown to be one of the largest trees east of the Rocky Mountains. In 1876 it measured thirty feet in circumference, and its roots have long since absorbed the waters of the spring. Flushing finally passed out of the hands of the descendants of the Rodman family in 1875, after being continuously occupied by them for a period of one hundred and sixty-three years — a rather unusual history in this part of the world. Gilbert Rodman, son of William Rodman, of Flushing, and father of the subject of this memoir, purchased Eddington from the estate of his father-in-law, Richard Gibbs, shortly after his death in 1795. On this farm Dr. Rodman passed his early years. He was

educated at the Lower Dublin Academy and in Burlington, New Jersey; studied medicine under the late Dr. Joseph Parrish, and graduated from the medical department of the University of Pennsylvania in 1827. The subject of his thesis was "Cholera Infantum." Soon after his graduation he went to Moorestown, New Jersey, to take charge of the practice of Dr. Spencer, of that place, while he was absent in Europe. On the return of Dr. Spencer he settled in Bustleton, Philadelphia, and on December 31, 1828, he was married to Miss Martha Moore, daughter of Dr. John Moore, of Philadelphia. Three children were born to them—two sons and one daughter. The daughter alone survives. His wife died March 10, 1858. In 1832, finding a country practice too laborious, he removed to Philadelphia, settling on Ninth Street below Arch Street. He remained there for several years, rapidly acquiring a practice, and from there removed to the south side of Arch Street between Eleventh and Twelfth Streets; from there to 1103 Arch Street, and from there to 1127 Arch Street, where he resided until his second marriage, January 23, 1872, to Miss Deborah Kirkham, daughter of William Kirkham, a merchant of Philadelphia.

Dr. John Ruan, an uncle by marriage of Dr. Rodman and a Fellow of this College, who died in 1845, and of whom a biographical memoir was read before the College by Dr. Henry Bond, had at that time the largest obstetrical practice in Philadelphia. Dr. Rodman succeeded to the better portion of this practice, and for many years he enjoyed one of the largest and most lucrative obstetrical and general practices in Philadelphia. He was elected a Fellow of this College in 1843. He was a Censor from 1861 until his death, a period of nearly thirty years, and always took an active interest in its proceedings. After his second marriage, in 1872, he removed to 2008 Spruce Street, and then practically retired from his active professional duties, which he had been gradually reducing for a number of years. He was a consulting physician to the Preston Retreat for many years, and one of the managers of the Philadelphia Dispensary. The active and exacting duties of a laborious practice left Dr.

Rodman no time for the literary duties of his profession, and I do not know of any contribution which he made to medical literature.

From 2008 Spruce Street he removed to 2100 Spruce Street, where he continued to reside, with the exception of the summer months, which were passed at Egremont, the beautiful country seat of his wife, at Edgewater Park, New Jersey. After his second marriage, in 1872, he rapidly diminished his practice, but continued active and in the enjoyment of good health up to within a few months of his death. He had a prolonged and serious illness in the autumn and winter of 1885 and 1885, from which he recovered; but from that time the infirmities of age, which had already begun to declare themselves, gradually increased. His mind was clear, his spirits cheerful, his step elastic as of old; but in the winter of 1889 and 1890 he began to fail rapidly. There were several rallies for a short time, but there was a gradual weakening in spite of all that was done for him. On June 11, 1890, he was removed from his city residence to his country seat at Edgewater Park, New Jersey, and on the 20th of June he passed peacefully away, just eight days after he had completed the eighty-fourth year of his age. Dr. Rodman was a consistent member of the Protestant Episcopal Church, and for many years was identified with St. Stephen's, of which he was for a long time a vestryman. After his second marriage he transferred his allegiance to St. James's, and one of the last official acts of its late rector, the Rev. Dr. Nicholls, now Bishop of California, prior to his departure to his new diocese, was to officiate at his funeral. Dr. Rodman was a kind father, an affectionate husband, an exemplary Christian gentleman. He was actuated by high desires, noble impulses, and a profound sense of duty. Descended from a long line of worthy men, he filled a quiet, modest, unassuming place, but one none the less useful on that account. With him has passed away one of the strong links which bind us to a past generation of physicians, when medicine was practised more as a profession, and less as a trade. He was successful, not because he advertised him-

self, but because he was a gentleman by instinct and nature; because he was honest, sympathetic, devoted, and true; because his patients confided in him, and because he repaid their confidence with skill, fidelity, and unceasing attention. He was essentially one of the old-time family physicians, now fast disappearing from the public gaze, who were almost members of the families in which they practised. They possessed the confidence of all the members of the family; attended the mothers in confinement, the fathers in illness; vaccinated the children; brought them successfully through the trials, tribulations, and dangers of infancy and childhood, from lancing their gums to the establishment of full adolescence; attended the weddings, were present at the funerals, and were the chosen friends, counsellors, and confidants of the whole family. The most delightful associations of my professional life are connected with families which he had attended for years, and to which I was introduced by him, and some of which he had inherited from his uncle, Dr. John Ruan.

The advent of modern specialism, which requires a different physician for each case of illness in the family, has done away with all that, and the old-time family physician is rapidly becoming a memory of the past. Let us hope it is better for the patient. It is certainly better for the specialists. Of Dr. Rodman it may be truthfully said that he filled an unostentatious but useful place in the battle of life. An active practitioner for half a century, the world was better for his having lived, and many a patient to-day respects and reveres his memory.

DISCUSSION.

Dr. Alfred Stillé: I was drawn to this meeting by my desire of hearing the memoir of Dr. Rodman, which has just been read, and which has in every respect gratified me. Dr. Rodman was at the time of his death the oldest Fellow of the College. In various ways, personally and officially, Dr. Rodman and I were thrown together for a number of years. In the days before the war, as you may remember, Mr. President, although your memory does not go back so far as my own, there were frequent social meetings of the profession. As I have often remarked, there was hardly an evening

in the week, from Monday to Saturday, when there was not a meeting which was almost exclusively medical. On Monday evening there was the club, composed of the survivors of an old editorial corps, of which Wood and Bache, Laroche and Bell, and Coates and Bond were members. Then there was a Tuesday Evening Club, and a Wednesday Evening Club, and the Thursday Evening Club, to which I had the honor of belonging from its foundation until its dispersion during the Civil War, when, after various attempts at its resuscitation, it died a natural death. On Friday evening the Tea and Coffee Club, of which Dr. Rodman was a member, held its meetings. On Saturday evening we had the Wistar Party. At all of these social meetings our friend Rodman was pretty sure of being found. His unobtrusive manner, the quietness of his tone, and the moderation of his opinions upon all matters which happened to be the subject of conversation, at once distinguished him from many of his contemporaries, and especially from the younger members of the profession. He was modest among the modest in every sense of the word—modest in his indisposition to proclaim his own title to anything of knowledge, or work, or whatever it might be, and modest in his demeanor and conversation, always keeping rather in the background than coming into the full light. He was, above all, an upright man. I do not suppose that any human being ever rightfully accused him of a misstatement or an unkind word.

Thus he passed through life, and I may say that I always regarded him during his career as a model of what a practising physician should be in his professional and social intercourse. That he did not take a conspicuous part in the public affairs of the profession was, I think, owing chiefly to that modesty upon which I have ventured to dwell; but he was none the less esteemed for the virtues which he conspicuously possessed.

I remember during the illness which occurred some years ago, and which was referred to in the biographical notice, going to his bedside when he was well enough to see his friends, and thinking to myself as I left the house that none but a man of so tranquil a temperament, and who was at such perfect peace with himself and all mankind, could have survived the attack which he overcame. There was nothing nervous, excited, or excitable about him. I remember that on that occasion he seemed to me, knowing through what straits he had passed, to have made a perfect resurrection. Yet I think that he never entirely recovered, either in appearance or in strength, from the shock he then received, so that when he finally succumbed in the attack which deprived the profession of his serene presence, his death was looked upon as the peaceful sleep that in the course of nature comes to those who have reached the evening of life.

A CASE OF ACROMEGALY.

By CHARLES W. DULLES, M.D.

[Read October 5, 1892.]

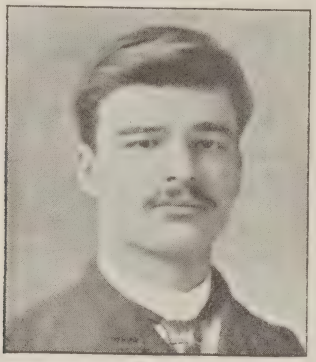
The subject of acromegaly was brought before the College last January and May, when Dr. F. A. Packard read a paper on this subject and a disease that simulates it (ostéo-arthropathie hypertrophiante pneumique—Marie), and Dr. Dercum exhibited two patients with acromegaly. Since that time, to confirm the commonly entertained opinion that an unusual occurrence is likely to be soon followed by others of the same sort, a case of acromegaly has unexpectedly presented itself to me.

On July 7th, in the house of a Syrian client, I was asked to examine and treat a friend who was newly arrived from Constantinople. The following account describes what I found:

A. P., born in Turkey in Asia, twenty-seven years of age, unmarried; height, five feet seven and a half inches; weight, about 160 pounds; has a good family and personal history, having lost two brothers in infancy and having five now living and healthy, and both parents living. Six years ago he was in this country, doing business in Chicago. As the accompanying photograph (Fig. 1), taken at that time, shows, he was then a well-formed and healthy young man. After this he went to Constantinople to carry on the Oriental part of a business that consisted in buying rugs, etc., and sending them to this country to be sold. Owing to involvement in the affairs of those for whom he acted, he was accused of violating the laws of the Ottoman Empire, and on March 4, 1890, was cast into a Turkish prison, where he remained for about eighteen months—until August 27, 1891. On leaving the prison he had no business, and was under a cloud of distrust and depression until he left Turkey to come to America, where he arrived fifteen days before I saw him.

While he was in prison his hands and feet began to enlarge, so that everybody said that he was getting fat, and his shoes (sandals) became too small

FIG. 1.



for him. About six months ago, he says,[1] he began to be oppressed with drowsiness, especially after meals, when he could hardly speak, but had to

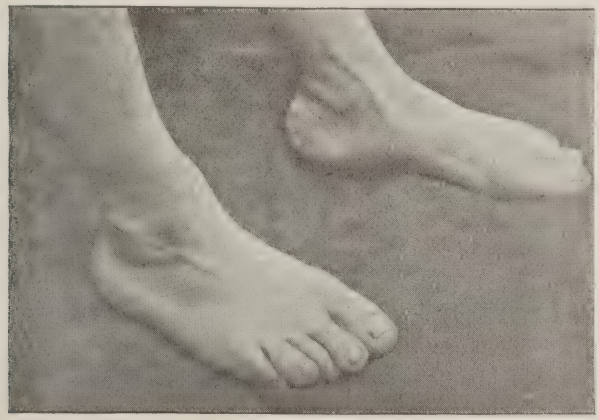
FIG. 2.

lie down and take a nap. For about three months he has had profuse sweats.

[1] The periods are not quite trustworthy because the patient does not seem to remember dates accurately.

When I first saw him he presented an appearance of melancholy, and a face that at once suggested to my mind the condition described by Marie as "acromegaly," and what appeared in the patients exhibited to the College last May by Dr. Dercum. The second photograph (Fig. 2) brings out the peculiar, massive, leonine countenance, the broad forehead, the wide malar eminences, the large mouth, the broad lower jaw, the large chin, the thick lips, the transverse wrinkles of the forehead, which make up the facial picture of acromegaly, and which are in marked contrast to the face in the photograph taken six years ago. Figure 3 shows the feet. In addition to

Fig. 3.



what the photographs show, it may be stated that there is lateral projection of the rami of the jaw, which is also enlarged, especially in the transverse direction. The form of enlargement generally seen in acromegaly seems to be an increase in the perpendicular and antero-posterior measurements, so that the teeth of the lower jaw overlap those of the upper, and the face is of an oval cast. In the present instance the lower jaw seems to be enlarged, in common with the whole of the bones of the face, except that its breadth is disproportionate. Standing behind the patient at a distance of a few feet, both rami are plainly seen extending behind the lines of the neck. This is not the case in normally formed individuals, in whom the rami of the jaws are not visible, or only barely so, when looked at from behind the subject. The wideness of the lower jaw is conspicuous on putting the fingers into the mouth and passing them between the upper maxilla and the ramus of the lower jaw, in which situation the coronoid process can be plainly felt quite outside of the superior maxilla. From the outside the articular process of the lower jaw can be seen and felt through the auditory meatus, sliding far

forward when the mouth is opened, and moving back as it is closed. The lateral enlargement of the lower jaw caused the lobes of the ears to protrude and the lower part of the ear (tragus and anti-tragus) to stand further from the middle line than the upper parts of the concha. The teeth of the lower jaw are somewhat separated, but they fall within the line of the upper teeth.

The hands and feet are characteristic. The former are large, and have the "sausage-shaped" fingers described by several writers. The feet are very large, and have the fleshy pad along the outer side, which is spoken of as peculiar to this disorder. There is also a thick pad under the os calcis and somewhat in advance of it on the sole of the foot.

A careful physical examination did not disclose any evidence of organic lesion of any of the thoracic or abdominal viscera. The urine presented no peculiarities on chemical and microscopic investigation.

The eyes were examined by Dr. George E. de Schweinitz, whose friendly interest in the case was increased by the fact that he has already examined the eyes of other persons suffering from acromegaly. Dr. de Schweinitz reports his findings as follows:

"The optic disc of the right eye is irregularly oval, of normal color, having at its nasal side a slight crescent. The fibre-layer of the retina in the neighborhood of the optic papilla is slightly hazy. There is a faint general absorption of the pigment-epithelium. The central vessels are normal in size and carry naturally-colored blood.

"The optic disc of the left eye is a vertical oval; its nasal side is bounded by a pigment-line and its temporal side by a sharply marked greenish border. Otherwise the conditions are the same as in the right eye.

"In the right eye there is compound myopic astigmatism; 1 D. of corneal astigmatism with its axis horizontal. In the left eye there is simple myopic astigmatism; 2.50 D. with its axis at 15. With correction of the refractive anomaly:

O. D.—2 ⊃ 1 cyl. ax. H. 6/IX.
O. S.—2.50 cyl. ax. 15 6/IX.

"The pupils are round, equal in size, and react to the changes of light and shade, convergence and accommodation. The pupillary reflex is more marked when a beam of light is thrown upon the right side of each retina than when thrown upon the left, but the hemiopic pupillary inaction (Wernicke's symptom) is not present.

"There is no paralysis of any external ocular muscle. The fusion power is good, and there is esophoria (insufficiency of the external recti) of 2 degrees.

"There is *typical left lateral hemianopsia*, with great contraction of the preserved field of vision, the contraction being the greater upon the right side. The dividing line between the dark field and the preserved field passes slightly in advance of the fixation-point. The color perception is normal in the central area, and there is a normal sequence in the appreciation of the colors in the area of preserved vision.

"The interesting feature of this case, so far as the ocular symptoms are concerned, is the presence of left lateral hemianopsia—a hemianopsia, moreover, that is probably due to a lesion back of the primary optic centres, inasmuch as there is preservation of the function of the sensori-motor arc of the pupil and the absence of Wernicke's symptom. The lesion would seem to be located in some portion of the visual tract posterior to the centres just named. Ordinarily in hemianopsia, when there is concentric restriction of the remaining half-fields, this is greatest in the eye opposite to the lesion. In the present instance precisely the contrary condition obtains, the greatest restriction being found upon the right side, while the character of the hemianopsia (left lateral) shows that the lesion that creates it must be upon the right side of the brain. Independent of an organic cerebral lesion, it is perfectly possible that a hemianopsia might be present under the influence of hysterical manifestations. A number of such cases have been reported. In several of the cases of recorded acromegaly, bi-temporal hemianopsia has been present. This, then, is peculiar in being an example of left lateral hemianopsia associated with this affection. It is quite possible that it is purely an association, and has nothing to do with the disease itself."

From this report it will be seen that the patient has a form of hemianopsia that is peculiar, in that it is homonymous, whereas in acromegaly the hemianopsia is usually bi-temporal.

Taking the picture of acromegaly drawn by Marie, we find this man's case to resemble it, in that there have been depression of spirits, profuse sweating, intense thirst, voracious appetite, great lassitude and drowsiness, especially at and after meals, severe and protracted headaches and marked melancholy of disposition, together with the enlargement of hands, feet, and face that are considered to be peculiar to this curious ailment.

Acromegaly is by no means as rare a disorder as might be supposed from the fact that only a few cases have come to the notice of medical men in this country. The often-quoted essay of Dr. Pierre Marie and of Dr. Souza-Leite (London, New

Sydenham Society, 1891) contains brief accounts of forty-eight cases gathered from various authors, the earliest being that of the French surgeon Saucerotte, published 116 years ago (1776), and Dr. O. T. Osborne, of New Haven, in an interesting report of a case he had studied (*Am. Journ. of the Med. Sciences*, June, 1892), speaks of it as the eighth case reported in the United States.

The reasons for considering this a special disorder, and the differences between it and osteitis deformans and leontiasis ossea, are fully discussed by Marie and his pupil Souza-Leite, and will, I think, satisfy any candid reader. According to Marie, the peculiarity of acromegaly consists in a marked enlargement of the bones and of the overlying tissues in the hands and feet, and also in the face, while the bones of the skull and of the trunk are usually unaffected. The enlargement of the bones causes a typical appearance of the face and of the extremities. The visible changes seem in many cases to be the consequence of profound distress or depression, and they are accompanied by lethargy of mind or even melancholy, with drowsiness, headache, extreme thirst, and sweating. In a number of cases hemianopsia has been observed. This curious manifestation should be looked for in every case until enough cases have been investigated to establish the importance to be attached to its presence as a symptom, and the exact form of it. As already stated, the case that I have described presents a form of hemianopsia which differs from that which has heretofore been described. The case differs also in the mode of enlargement of the lower jaw, and these very differences make it the more desirable, I think, to put the case on record; for the literature of the subject is still very restricted, and it may be that with a larger material for study, there may be some modification of our ideas as to what is essential to the disorder.

An interesting contribution to this literature is an essay[1] supplied by Professor Mosler to that remarkable monument in

[1] Ueber die sogenannte Akromegalie (Pachyacrie).

honor of Virchow's semi-centennial celebration, the *Internationale Beiträge zur wissenschaftlichen Medicin.*

The patient whose case I have described was sent by me to Dr. Packard and to Dr. de Schweinitz for examination, and would be before you to-night but that he wearied of treatment in a hospital, and left the city.

The treatment to which the patient was subjected was directed chiefly to the digestive tract, which was much out of order. An obstinate constipation was broken up; a suspicion of lumbricoids (a very common affliction of Turks and Syrians) was followed up until it proved ungrounded, salol and phenacetin were given for headache, and strychnine was given as a general and nerve tonic. During the month in which the man was under my care he improved in some respects; but the improvement was solely in regard to the state of his digestive apparatus and the state of his mind. At times he acquired a certain degree of cheerfulness, but this did not last long. On leaving the city for a short summer holiday I had the patient admitted to a hospital, and on my return I found that he had decided to leave it, and that he had also left the city.

A CASE OF ACROMEGALY.

By SOLOMON SOLIS-COHEN, M.D.

[Reported October 5, 1892.]

In connection with the paper of Dr. Dulles, I desire to present photographs of a case of acromegaly that I accidentally met with. The subject is not aware that he has any disease, and, while he has kindly given me permission to publish his measurements and photograph, he has stipulated that the face should be rendered unrecognizable.[1] I saw this man attempting to play foot-ball, and was amused by the way in which the big, burly fellow was literally played with by a much smaller man. He is a native American, and born of native-American parents, of English stock. He is six feet two and a half inches in height, and apparently of great muscular development. At the age of sixteen years he weighed one hundred and eighty pounds. His present weight, at the age of twenty-five years, is two hundred and thirty-eight pounds. Up to four years ago he was a blacksmith, and was then able to wield a thirty-pound sledge. Now he cannot lift much weight, and cannot vie with smaller men in athletic exercises of any kind. He is clumsy and sluggish in movement, and although he can run long distances (four miles) without becoming "winded," he is quite slow.

He has not spontaneous or intense headache, though it may be brought on by close study; occasonally he has confusion of ideas. He claims to have a remarkable memory for facts in history and literary quotations, but he finds difficulty with mathematics or other studies requiring deep thought. This is not necessarily abnormal.

He has periods of drowsiness. His appetite is exaggerated. He has not noticed excessive thirst or polyuria. A single examination of the urine failed to show albumin, sugar, or tube-casts.

I have here tracings of the hands in which the characteristic thickening and dumpiness of the fingers is quite noticeable. The hands (Fig. 1) are enlarged laterally rather than longitudinally, having a characteristic spade-

[1] This has been skilfully accomplished by retouching the photographs prior to reproduction. The untouched photographs were exhibited.

shape. The soft parts as well as the bones are involved. The fleshiness is marked; the skin is thick and tough, and the fine lines almost obliterated, while the main furrows are coarse and deep. The segmentation of the fingers is thus quite marked in the photograph. The nails are short and broad, not striated. The grasp is not strong. I was able to endure without wincing his greatest pressure upon my own hand.

His feet are broad, flat, large and fleshy, and he has had to increase the size of his shoes, more particularly in breadth, within two years.

Fig. 1.

Fig. 2.

In two years the size of his hat-band has twice been increased, from six and three-quarters to seven, and now to seven and a half. His nose and ears have been large since boyhood, but his nose has lately much increased in size. Three years ago he first noticed the increase in the size of other features and changed expression of his face, and at his last vacation his parents noticed that in the nine months of his absence from home his physiognomy had altered considerably. He thinks that at the age of sixteen years he was as tall as at present. His father and brothers are tall men, but their noses and hands,

the man distinctly states, are unlike his. They do not stoop. Very noticeable in his attitude on the foot-ball field was his stoop forward—the cervico-dorsal kyphosis, with tendency to forward thrust of the head. This is shown in one of the photographs. (Fig. 2.) Negatively it is shown in another photograph (Fig. 3) by the manner in which he has thrown the trunk backward from the waist in order to get the head into a vertical position. The marked enlargement and varicose condition of the veins of his legs also attracted attention. There is a slight paunching of the abdomen. The nose is large, thick, characteristic. The patient states that it is "growing." The superciliary ridges are prominent, the walls of the frontal sinuses much enlarged, giving a characteristic forehead, as well shown in the profile; the malar bones are enlarged, projecting laterally, so that with the vertical increase in the size of the lower jaw the outline of the face is the typical lengthened ellipse of acromegaly.

The ears are very large, standing out at right angles with the skull, and the cartilages are thickened almost to rigidity. The lips are thick and the lower lip overhangs. The tongue is broad, thick, deeply furrowed. The lower teeth do not project beyond the plane of the upper, although the chin is much enlarged vertically. The scapulæ are enormous, and the acromion and coracoid processes can be easily demonstrated, and the distance between them seems to be quite great. The sternal ends of the clavicles stand out markedly as knobs. The sternum is unusually long, and the xiphoid process is apparently ossified.

The thyroid cartilage is large, thick, and gristly, the cricoid is apparently ossified; internally the larynx is large, but presents nothing abnormal. Although the external nose is immense, the nasal chambers do not appear to quite correspond in size; but I did not make accurate measurements. The thyroid gland was not demonstrable, although careful search was made for it by Dr. Franklin H. Hooper, of Boston, and myself. Dulness on percussion over the upper portion of the sternum gives some color to the theory that there is a persistent thymus in some of these cases.

The thoracic and pulmonary development is remarkable. The ribs are long, the curves and intercostal spaces correspondingly exaggerated. There is an expansion of more than six inches, and the vesicular tone is perfect both on percussion and auscultation. The heart is apparently proportionate to the frame. No lesion was detected.

One feature present in the case that I have not seen mentioned in connection with other cases is the ease with which hyperextension of the forearm could be produced. Dr. Allis tells me that this, like the similar condition in anæmic girls, is an evidence of muscular weakness. The photographs show this, and also a similar phenomenon at the knees. (Fig. 3.) His patellæ readily slip out of place, and he has been laid up three times by luxation of the patella.

The rough examination that I was able to make detected no lesion of the

eye. I did not have an opportunity to have an ophthalmoscopic examination made.

The photographs do not adequately represent the case, but they are, I think, sufficient to verify my descriptions. The case, in connection with those

Fig. 3.

likewise "picked up" by Dr. Dercum, suggests that this affection is perhaps not so rare as one might be inclined to imagine. The man is young, and may be expected to develop other symptoms, while the present conditions will become more marked.

DISCUSSION.

Dr. F. A. Packard: The condition of the fields of vision in Dr. Dulles' patient, as shown by the examination of Dr. de Schweinitz, helps to prove that there are a large number of cases of acromegaly in which tumor of the

pituitary body, on which so much stress has been laid, has nothing to do with the etiology of this disease. An interesting observation has been made by Marie and Marinesco. They found that excision of the thyroid gland causes in rabbits increase in the size of the pituitary body. In the case which I had the pleasure of presenting to the College last spring, the thyroid gland was absolutely imperceptible to the touch. I have been thinking quite seriously, if the English experiments of transplantation of a thyroid gland to the peritoneal cavity in cases of myxœdema continue to prove favorable, of persuading my patient to try the effect of this, for he is willing to do almost anything that offers a chance of relief from his severe headaches. I think of this means of endeavoring to obtain relief for him, because (1) I believe that his headaches are due to enlargement of the pituitary body; (2) the enlargement of the pituitary body may be caused by ablation of the thyroid gland; (3) his thyroid gland is at least atrophied; and (4) experiments in England with transplantation of the thyroid gland of animals into the peritoneal cavity of man have met with success in cases of myxœdema, with which disease I believe acromegaly to be closely allied.

Dr. F. X. Dercum: The cases presented to-night constitute an interesting contribution to this subject. I am glad that Dr. Packard has come to the view that the pituitary body does not bear a causal relation to this affection. In neither of the cases which I reported was there headache, and in only about one-third of the reported cases has headache been noted. It appears to me that only those cases come under observation in which there is headache or eye trouble, and that many cases exist where the individuals have no occasion to consult a physician.

In connection with the pituitary body, I would call attention to the fact that some years ago Dr. Mitchell reported a case of cerebral aneurism which probably had its origin in an anomalous vessel of the circle of Willis. The whole pituitary body had been destroyed by the aneurism, and there had been bi-temporal hemianopsia, but there was no trace of any peculiar enlargement of the face, hands, or feet.

Dr. Cohen's case is also of interest, as showing that the affection is not merely an "acromegaly"—that is, enlargement of the ends of the body—for, in his case, as in one of mine, there is overgrowth of muscular tissue and of the bony walls of the chest. While there is a tendency for the accentuation of the enlargement in the ends of the body, it certainly is not limited to these parts, but may appear elsewhere.

The observation in regard to the hemianopsia is of exceeding interest, and brings up the whole question whether we are to regard the bi-temporal hemianopsia found in other cases as due to the enlargement of the pituitary body or not.

Dr. C. Dulles: I wish only to say that, in studying a rare disease there is always a little risk of making the mistake of considering that an example of the disease which really is not such. When I first saw this case I sent the

patient to Dr. Packard and to Dr. de Schweinitz, who had examined other persons with acromegaly, so that, if they had any doubts as to the diagnosis, they might express them when opportunity offered. There is some danger that cases of mere gigantic development may be considered as cases of acromegaly. If I understand Marie correctly, this disease does not consist simply in a mere overgrowth of certain tissues, but it is a disordered condition of the system, of which overgrowth of certain tissues is only one of the manifestations. Genuine instances of acromegaly have something more than this, and cases thought to be such ought (if medical terms are to have any fixed meaning) to present a resemblance in their clinical history to the picture drawn by Marie, who first described what he named "acromegaly." I will be candid enough to say that the history which Dr. Cohen has given, and the pictures which he has presented, do not impress me with the idea that his patient had acromegaly. In order to justify that diagnosis we should be able to contrast his present condition, either by good description or by pictures, with something quite different within a recent period. The man is naturally large, has been a blacksmith, and is well developed as an athlete. The fact that he has a large head, legs and hands, and a curved back, does not justify the belief that he has what Marie called acromegaly.

Dr. Dercum refers to the small proportion of cases in which headache was present. My impression of the cases reported is that in them headache was a pretty constant symptom, and that where headache was not present there were other marked evidences of disturbance of the central nervous system, such as depression and melancholia, and that great lethargy was a distinct characteristic of this form of disease.

The photographs shown by Dr. Cohen strike me as showing a man who is pretty uniformly well-developed, and who has, what Marie says does not occur in acromegaly, an unusual development of the long bones. The hands look graceful and well proportioned. I do not think that these are the sausage-shaped fingers of Marie. I see nothing peculiar about the pictures except that they show a very large and muscular man, with unusually long arms.

In discussing a scientific subject it is important to be candid, and if one has doubts, to express them, in order that they may be set at rest or confirmed, as the case may be. In the case I have described I have shown you photographs comparing his present condition with that of six years ago, and have stated that, in my opinion, the patient's clinical history is that of acromegaly. He has been a sick man, with a series of symptoms which fill up pretty accurately the picture of acromegaly depicted by Marie and followed out by others; and therefore I think it proper to put him in this class. The peculiarity of the eye manifestations has been fully described, and others may now judge of the correctness of the diagnosis.

Dr COHEN: I am glad Dr. Dulles has raised this question. I have studied the case carefully from the standpoint which he has suggested. The photo-

graphs do not show the appearance so well as does the man himself, but I think that those who are familiar with the disease will recognize that this is the same physiognomy and the same hands as were seen in the cases presented by Dr. Dercum. The College must put some faith in my description. Big men and big noses are common, but not such men and such noses; curved backs, protuberant bellies, varicose veins are not rare; but these curves and varicosities are associated as parts of an uncommon whole. It is not any one feature of the case, but the *tout ensemble* that makes the diagnosis plain. I am sorry that my language was so faulty that it failed to impress Dr. Dulles that just the very points he lays most stress upon as negativing acromegaly are not present in the case, while just the very points I would lay most stress upon he omits to mention. The tracings and photographs of these hands are not normal, nor are they, to my mind, graceful. The deep furrows, the cylindrical shape, and thick, fleshy pads of the fingers, and the lateral enlargement of the whole hand, are marked, and had attracted the attention of the man's fellow-students. They had especially remarked that the wrists were not enlarged in proportion. Further, the face is striking, and the man himself reports a change in his features and an increase in the size of his hat. His ears, which were disproportionately large as a boy, are now not so much out of place. The projection of the skull over the frontal sinuses and that of the malar bones are marked; the lips are thickened, the lower lip overhangs, and the vertical depth of the jaw is increased. If to these facts we add the further fact that although large he is not, in any proper use of the the words, well developed, but that, as I carefully stated in the first place, he is awkward and clumsy, and although when a blacksmith he could handle a heavy hammer with ease, yet now, when he is apparently so much more muscular, he has little strength, I think that there can be no doubt that this is a genuine case of acromegaly. I should also draw attention to the apparent atrophy of the thyroid gland.

Dr. F. X. Dercum: From the photographs and the history, I have no doubt that the case reported by Dr. Cohen is one of acromegaly. The face has the vertical elongation, the vertical depth of chin, the increase of the malar prominences, and also the thickening of the superciliary ridges. The hands are almost typical. The man is still young, and in the course of a few years the case will probably become more pronounced. In the case reported by Virchow, which has never been disputed, the nose and mouth were not excessively enlarged, but the hands were large, and so also were the muscles.

I do not think that the psychic depression is a necessary feature. It was present in one of my cases for a short time and not present in the other. Neither of my cases had occasion to consult a physician. I really picked them off the street. Neither of my cases had headache, nor had they eye changes.

A CASE OF RAYNAUD'S DISEASE, ASSOCIATED WITH ANGINA PECTORIS.

By RICHARD A. CLEEMAN, M.D.

[Read October 5, 1892.]

The case which is recorded below possesses several features of much interest, but it is reported chiefly on account of the supervention of angina pectoris in the course of that complex of symptoms known as Raynaud's disease.

The patient was sixty-two years old at the time of his sudden death; he was never married, and had devoted his life to mercantile pursuits. He was naturally of marked sanguine temperament, of strong muscular development, and high animal spirits. Though enjoying in general rugged health, he suffered through his whole life, though to a less degree after middle age, with bromidrosis of the feet. Besides the tendency to excessive perspiration, there were times, especially at night, when the skin of his feet would become actively congested; an itching so intense that he could not refrain from scratching. He would scratch till a little blood would flow, when the condition would pass away; there was no pain and no observed relation to the exercise of walking, as in the erythromelalgia of Mitchell; it seemed a simple and fugitive irritation of the vaso-dilator nerves of the parts.

When a boy he had an attack of typhoid fever, during which he suffered from epistaxis to an alarming degree.

At twenty-one years of age, while passing through the tropics, he contracted the severe form of malarial fever known as "Chagres fever." The attack was a serious one, and many months passed before he felt himself quite well again. But he apparently entirely recovered, and for more than thirty years enjoyed exceptional health, disturbed only by the discomfort of the feet above described and the occasional slight ailments due to a rather free life.

After he was more than fifty years old he had occasion to remain several hours on a river wharf exposed to a damp, cold atmosphere, and became

thoroughly chilled. From this time, without obvious disease of any organ, he lost his strong vigorous appearance, became irritable in temper, very sensitive to cold, and indisposed to physical exertion. From this condition he recovered very slowly; indeed, he never regained entirely his weight and activity, though, perhaps, this was scarcely to be expected at his age.

Now appeared the symptoms so graphically described by Raynaud (M. Raynaud, *Nouv. Dict. de Méd. et de Chirurg. Prat.*, p. 636, vol. xv., 1872). One or more fingers or parts of fingers of both hands, especially on cold days, would become pale, waxen-looking, shrivelled, and cold, the well-known *digiti mortui;* or occasionally this took place in only one hand; there was, as a rule, no pain, and the parts would after a variable time return to their normal condition. Rarely, however, the morbid condition progressed beyond mere "local syncope," and superficial gangrene of the integument over the pulp of a finger resulted with subsequent painful ulceration. The paroxysms were more frequent in winter, but they also occurred in summer, especially in damp weather. At the time of his death the patient had been subject to these attacks at least six years.

On a winter's day, without any apparent exciting cause except the low temperature, which, however, was not excessive, the patient was suddenly seized with an agonizing pain, referred to a point behind the upper part of the sternum, and seeming to radiate down both arms as far as the elbows; his face wore a look of extreme anxiety, he appeared afraid to breathe, his pulse was feeble and wanting in tension. This attack of angina pectoris was exceedingly severe, lasting two hours. The patient said he had never suffered anything like it before, except that he had felt on several occasions a momentary pain behind the sternum, but of so transient a nature that it had disappeared almost the instant he was conscious of it. The angina was followed by a good deal of prostration, but after two days' rest the patient attended to his business affairs as usual. A week later, during very cold weather, he was found dead in his bedroom, having died apparently in the act of undressing for bed, and most probably from another attack of angina.

During this last week the peripheral vasomotor disturbances had continued to manifest themselves. One night the patient was awakened by a feeling of intense numbness in one of his fingers, the numbness being succeeded by a severe burning pain. No autopsy was made.

In reviewing the features of this case, it is interesting to note the development of the symptoms of Raynaud's disease in a man more than fifty years old, and their recurrence till the time of his death at sixty-two years of age; Raynaud having told us that four-fifths of these cases are to be found among females, and that the disease is so rare beyond thirty years of age that it might properly be called "juvenile gangrene;"

while M. Allen Starr (Pepper's *System of Medicine*, p. 1261, vol. v.), referring to the vulnerable age, says that only two cases have been recorded in persons fifty years old.

In this case there was probably a congenital predisposition to irritation of the vasomotor centres, as manifested in the habitual hyper-secretion of the skin of the feet and its periodical congestions, and also in the profuse epistaxis under the influence of the typhoid-fever poison.

The researches of Petit and Verneuil (*Revue de Chirurgie*, 1883, "Asphyxie Locale et Gangrène Palustres," pp. 1, 161, 432, 699), following the lead of Moursou, Duroziez, and others, which seem to discover a close relation between Raynaud's disease and malarial poisoning, make the "Chagres fever" of the patient of great significance. A recrudescence of the poison in his declining years, due to the exposure on a river wharf, at once suggests itself as the cause of the "local syncope" and gangrene.

But what of the angina pectoris? Was this a part of the same vasomotor disturbance, or was it a mere accident in the case—a coincidence only? Writers on Raynaud's disease do not refer to any special liability to the development of angina pectoris, though Petit and Verneuil (*op. cit.*, p. 24) record two cases in which angina accompanied that affection. They make no remarks, however, upon the association, except to deplore that the condition of the heart is not more often mentioned. Neither of these cases was fatal. On the other hand, among the clinical features of angina pectoris we do not find "local syncope" and its terminations described. It is true that Landois and also Nothnagel have described a variety of angina which they call *angina pectoris vasomotoria*, but the symptoms are different from those of the more common type to which this case seemed to belong. In the former there appears to be a general spasm of the peripheral arteries and that the heart is affected secondarily through the resistance offered to the blood-current by the contracted vessels, while in the latter the heart seems to be involved more directly. Is it being too speculative to attribute the angina in the case before us to a

spasm of the coronary arteries of the heart, or their branches, causing ischæmia of that organ just as the contraction of the digital arteries produced a local syncope in the fingers? The ischæmia would affect the ganglionic masses in the substance of the heart, affiliating this case with the first of the types in Eulenberg's classification of angina pectoris—the cardial excitor-motor form. Perhaps this is the mechanism of most cases of angina, and Cahen was right when, long ago, he placed the seat of the disease in the vasomotor system. It was always difficult to imagine a spasm of the heart-muscle itself, as supposed by Heberden and others, while the pulse was beating regularly or even violently, as occurred often in cases of angina.

DISCUSSION.

Dr. H. C. Wood: Raynaud's disease is to me one of the most mysterious diseases with which I have met. The theory which attributes these changes to vasomotor spasm and dilatation affecting the arterioles does not, to my mind, account for the phenomena of the disease. One of the most pronounced phenomena is the intense congestion which follows the period of contraction and whiteness. Now dilatation of the arterioles would not produce venous congestion. There must be something more than vasomotor disturbance to account for the intense lividity and venous congestion that follow the period of spasm.

I have seen one case which is of interest in connection with the case reported by Dr. Cleeman. This case long ago convinced me that other vascular territories than the periphery of the body may be affected with the series of changes which produce Raynaud's disease. This man suffered atrociously with fairly typical Raynaud's disease, lasting over months and years. The peculiarity of the case was that he also had attacks of localized headache, in which the pain in the head was similar in character to the pain in the fingers. The attacks of headache sometimes accompanied and sometimes apparently replaced the attacks of pain in the fingers. Most careful study failed to show any cause for the headaches. I have no doubt they were due to localized meningeal congestion similar to that which occurred in the fingers. What is the pathology of Raynaud's disease I think is not at all clear. As to treatment, I have found nothing valuable. The cases that I have seen have drifted into hopeless opium or morphine eaters.

Dr. J. Madison Taylor: As of interest concerning the question of treatment of this disorder, I might mention incidentally the case of a lady who came under my observation recently. She was married; about thirty years of age; had three very healthy-looking children. The malady began, I believe, about three or four years after marriage, and was chiefly manifested by excessive and constant pain in the fingers of both hands. She had consulted no end of physicians both here and abroad. Prof. Charcot, of Paris, prescribed antipyrine in large doses, up to sixty grains a day. This was most efficacious, and had produced no noticeable blood dyscrasia as yet.

Dr. S. Solis-Cohen: While I cannot from personal experience discuss cases of Raynaud's disease progressing to gangrene, I have seen three cases of less severe type, one in Dr. Da Costa's service and one in Dr. Bartholow's service at the Jefferson Hospital while I was chief of clinic there, and one in my own service at the Polyclinic recently, in which nitro-glycerin seemed to do good. At the Jefferson Hospital, arsenic was associated with the trinitrin in one case and iron in the other. The cases passed from observation too soon to say whether or not the benefit was permanent. In my own case nitro-glycerin and iron were employed, and the patient apparently recovered, and has remained well for the past six or eight months. This case, which I have elsewhere reported, likewise presented enlargement of the thyroid gland, a venous hum in the neck, slight anemia, menstrual irregularity, a tendency to anomalous eruptions, and a tendency to attacks of tachycardia, thus relating the case to Graves's disease.

What has struck me in studying these cases, as also in cases of exophthalmic goitre, in cases of angeio-neurotic œdema, in some cases of myxœdema, and in some cases of lithæmia, is that there seems to be a vasomotor ataxia rather than vasomotor paralysis or spasm. I have little doubt that in Dr. Cleeman's case the angina pectoris was an essential and not an accidental occurrence. It seems to me that there must be some profound nervous disturbance underlying this affection, some disturbance of the centres of organic life and nutrition. Just what it may be our data are too incomplete to enable us to determine, but the principal feature, so far as the vessels are concerned, seems to be rather a want of regulation of calibre than either a contraction or relaxation. In exophthalmic goitre the paralytic condition predominates, but at times there are spasmodic phenomena; in Raynaud's disease the spasmodic condition predominates, although there are times when relaxation is noticeable. Possibly the venous congestion may be explained by subsequent paralysis from reaction following the spasm, the want of control permitting the blood to accumulate in terminal veins from want of proper tone in the peripheral arterioles to pass it on. This is, I believe, the current doctrine.

Dr. Cleeman's case, and the cases he cites suggest that there may be a toxic origin for the various vasomotor disturbances. As in myxœdema, the want of the nutritive or protective action of the thyroid seems to induce the

peculiar mucoid degeneration or deposit associated with peripheral blushing and sweating, or, as in acromegaly, we have a condition of disturbance of the vessels with sweating, congestive headache, and polyuria, and other trophic disorders, which may be referable to a deprivation of the action of the thyroid gland, so here there may be some toxic element as the original causative agent. Thus the occurrence in cases of uric acid diathesis of angina, of circumscribed œdema, even of transient hemianopsia and blindness, as I have seen in a few cases, and of similar paralytic or spasmodic phenomena; the connection sometimes noted between rheumatism or chorea and Graves's disease; the presence in Graves's disease of thyroid enlargement and of menstrual disturbances, are similarly suggestive. I do not mean to say that these diseases are due to a common cause, but that they have in part a common mechanism. Similarly the urticaria that in susceptible subjects results from cinchonism or from the effects of certain articles of diet, suggests toxæmia as a part of the mechanism. What I wish, then, to call particular attention to is that there is a large group of diseases, involving many conditions that apparently have superficially no relation to each other, in which there is this want of control of the circulation, probably due to some defect in the organic nerve centres. This may arise from the action of toxic agents introduced from without, or resulting from perverted metabolism; or it may itself be the cause of metabolic aberration and be associated with other trophic changes. I have seen it in cases of diabetes mellitus. In my limited experience, in the spasmodic conditions, nitroglycerin with arsenic or iron seemed to do good, while in the condition where paralysis predominates, picrotoxin, as suggested by Dr. Bartholow, strychnine, digitalis, or other vascular tonic, does good.

In some of the cases I have classed in this group the appearance of blood-pigment or blood in the urine has been noted by various observers. I am not aware, however, of any publication, except my own, in which attention has been called to the occurrence of red blood cells in the urine (hæmocyturia) without macroscopic alteration. I have learned to search for this in all of these cases of vasomotor disturbance and to attach some diagnostic significance to it.

Dr. Taylor: With reference to the question of blood in the urine in these cases, I would say that in one case of exophthalmic goitre, blood was found to a marked degree in the urine on three separate occasions. I have seen the urine slightly bloody in one or two other instances, and smoky several times.

I may be permitted to say a word in regard to the treatment of these vasomotor ataxias, or whatever they may be called. One case of exophthalmic goitre, which had progressed for sixteen years without relief, seemed to be controlled by rising doses of hydrobromate of hyoscine, which first was given simply to relieve headache. It has relieved this and the irregular vascular phenomena and the extreme restlessness most satisfactorily. The skin, which had been very leaky for years, became normal again, and a great gain

in strength was noted, and now continues after the lapse of a year. This same substance has proved useful in other instances.

Dr. H. C. Wood: I doubt whether this affection is primarily a disease of the vasomotor system. No vasomotor spasm that we can produce artificially will cause the intense whiteness of the waxy fingers of Raynaud's disease, and no vasomotor paralysis that we can produce will cause the intense local congestion of Raynaud's disease. You may divide the nerves going to a limb, but you do not get a congestion like that of Raynaud's disease. It seems more probable that all the phenomena are trophic or there is a simultaneous disturbance of the tissues and of the bloodvessels; so that the disease does not belong among the pure vasomotor neuroses but among the trophic diseases. This would account for the rapidity of the ulceration. You cannot ordinarily produce ulceration in the fingers and extremities by the section of nerves.

Dr. Charles W. Dulles: I have had an opportunity to see what these waxy fingers look like inside as well as outside. Some years ago I amputated part of a finger of a patient of Dr. Musser, who had typical ulceration at the end of the fingers, as the result of Raynaud's disease. The incision was almost like cutting through paraffine, and there was no vessel that required ligation.

Dr. James Hendrie Lloyd: I should like to suggest here a possible analogy between Raynaud's disease and the disorder occurring in syringomyelia described by the French physician Morvan, and to which the name Morvan's disease has been given. It is a form of whitlow or analgesic felon slowly destroying the phalanges. In these cases the trophic disorder is supposed to depend upon neuritis consecutive to the peculiar degeneration of the cord known as syringomyelia.

Dr. Cleeman: Arsenic seemed to be of decided service in toning up the nervous system and rendering the attacks less frequent. The urine was not examined for blood-corpuscles.

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TWO CASES OF ACROMEGALY; WITH REMARKS ON THE PATHOLOGY OF THE AFFECTION.

By F. X. DERCUM, M.D.,
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[Read May 4, 1892.]

Inasmuch as the subject of acromegaly is attracting such widespread attention, a report of additional cases, especially if they present unusual features, cannot fail to be of interest; and further, while the disease is rare, it is probably more common, as these cases would seem to show, than is generally supposed. As a rule, only those cases come under observation which apply to physicians for the relief of some painful or distressing symptom. On the other hand, it is extremely probable that others, if not indeed the larger number, pass through life without the knowledge that they are the victims of a mysterious disease, realizing only that Nature has made them big of limb and feature and unpleasant to look upon.

Neither of the cases about to be described presented himself as a patient. Neither had had any occasion for medical advice since childhood. Both were discovered by accident, one on the street, the other in his workshop. Both proved willing subjects for study. It seemed to be a source of satisfaction to them to learn that their peculiar appearance was not a thing normal to them, but the result of a disease; and this may, after all, have been a very natural feeling.

Case I.—J. T. M., aged fifty-six years; married; a cutler by occupation, and a native of American parentage.

Family history: His father died at sixty-seven years, of heart disease; his mother at seventy, of some affection which he does not remember. He

had, in all, five brothers and two sisters. Of these, two brothers died in childhood, while a third appears to be in delicate health. The others, inclusive of the two sisters, appear to be well. He himself has a son of twenty-two and a daughter of thirteen, neither of whom present anything abnormal. He is further very positive in his assertion that no one of his relatives were ever affected as he is, and that they were all people of average size.

Personal history: As a child he had measles, scarlet fever, and whooping-cough, but since that time he has had no illness of moment. However, he used to take cold rather easily, and has occasionally had rheumatic pains. Nothing further could be elicited until finally he admitted that in former years he had suffered very much from emotional depression. He dated the

FIG. 1.

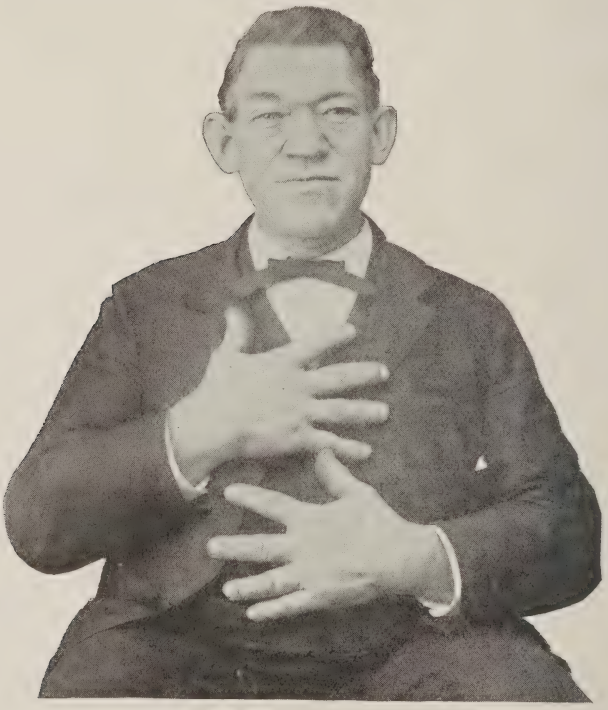
S. T. M., aged twenty-seven years.

origin of this trouble from his eighth year, when, upon one occasion, he had been very severely whipped for a fault of which he had been entirely innocent. He appears to have been an unusually sensitive child, for the unjust punishment to which he had been subjected made, in spite of all attempts at reparation by his parents, an indelible impression upon him. To use his own words, he "fell into darkness and worry," and as he grew older "the darkness and worry grew with him." When twelve to fifteen years of age

his depression deepened at times so much as to resemble melancholia. He used to be "so sad and heavy-hearted" that he could not sleep, and lay awake at night "worrying about things that he ought not to have done." This mental state persisted off and on, though in a lessened degree, throughout youth and adult life, up to some fifteen years ago. Since then he has felt quite at ease and even cheerful.

When a young man he appears to have been quite slender and slight in build, as the accompanying photograph (Fig. 1), taken when he was twenty-seven years of age, seems to show. He did not notice any marked increase

FIG. 2.



S. T. M., aged fifty-six years.

of any part of his body until about middle age. Indeed, the onset of his symptoms was so gradual that he cannot fix the time when it began, but he remembers very distinctly that it was only ten or twelve years ago that he realized the size of his hands was very great. Since that time they have continued to grow larger. He has suffered neither from alcoholism nor syphilis.

Present condition: As seen by the accompanying photograph (Fig. 2), the face and hands are excessively large. In the face the features show marked increase in size as compared with Fig. 1. The supra-orbital ridges, the nose, the malar bones, the chin, and even the ears are striking evidences of this. The mouth, too, looks like an enormous transverse slit, while the tongue when protruded is so large that when thrust downward the patient can almost cover with it the hypertrophied chin, and when thrust upward fully the lower third of the nose. An examination of the mouth reveals that he has lost many of his teeth, and further, that the lower jaw projects far in advance of the upper. The face and mucous membranes are rather pale.

The hands are excessively broad and flat while the fingers look blunt and stumpy, while the natural furrows of the parts are much exaggerated. The nails are broad, flat, and short, and broken off at the ends as though brittle. They also present in varying degree a fine longitudinal striation. The wrists do not seem to be enlarged in proportion to the hands. The feet show an increase in size corresponding to that of the hands. They are also excessively broad and flat, while the nails are similar to those of the hands in appearance.

The following measurements were made:

	Right. Inches.	Left. Inches.
Length of hand from wrist to tip of middle finger	8½	8½
Circumference of hand at knuckles	10	10¼
" " metacarpus, including thumb	12	11¾
" " forefinger, proximal phalanx	3⅝	3⅝
" " wrist	8½	8⅝
" " forearm, junction of upper and middle third	11⅞	12
" " arm	13⅝	13⅜
Length of foot, heel to tip of great toe	11⅛	11¼
Circumference of foot at ball of great toe	11	10¾
" " " " instep	11¾	10½
" " " across instep and heel	14¾	15¼
" " ankle	9¼	9⅛
" " leg, junction of middle and upper third	15⅞	15⅝
" " thigh, junction of middle and upper third	22¾	22¾
Length of nose, root to tip		2⅜
From zygoma to zygoma		5
Depth of chin		2¼
Circumference of neck		15⅝
" " chest, at nipples, expiration		43½
" " " " " inspiration		45
" " abdomen		44

From these measurements it is evident that the face, hands, and feet are decidedly enlarged. As regards the arms and legs, the figures do not permit us to predicate any increase in size. It is very probable, however, that as regards the chest some increase has taken place. This is evidenced, not so

much by the figures, as by the appearance of the costal cartilages and the sternum, which are much hypertrophied.

As regards the length of the feet, which is excessive, it should be stated that it is mainly due to a marked increase in the tissue over the calcaneum, if not indeed to an increase of the calcaneum itself. The appearance is very striking, and is more marked than in the heel of the negro, which it forcibly recalls.

In addition, the thyroid gland is much enlarged, as are also both testes, especially the right, which is enormous, and recalls the corresponding organ in the sheep. The penis does not seem to have shared in the increase.

Dulness over and adjacent to the sternum, such as has been noted by Erb, and which is supposed to be indicative of persistence and enlargement of the thymus gland, was not present.

The patient suffers from general weakness. He is easily fatigued. There is, however, nowhere any local palsy, nor is there anywhere any loss or modification of cutaneous sensibility. The grip is diminished, and the knee-jerks are sluggish and lessened.

Stoops slightly, but there is no kyphosis.

Examination of the eyes is negative, with the exception that in the left eye the field is somewhat contracted in its temporal half. Smell is decidedly obtunded. Taste is apparently well preserved. Hearing appears to be normal.

The urine has a specific gravity of 1026, and is negative to all tests.

When questioned, the patient states that he rarely has headache; that he has an excessive appetite, and that he frequently eats "more than is good for him," also that he is always thirsty, that he drinks about a quart of water with each meal, and a great deal between; further, that he is very apt to fall asleep in the daytime, especially if he sits down, and that he will even fall asleep while people are talking to him. Thinks that his memory is impaired.

Further, he sweats a great deal, especially about the head and neck. He has but one symptom, however, which distresses him, and that is, that whenever he exerts himself a little his heart palpitates.

Sexual power is also much diminished; though in estimating the value of this symptom the patient's age must be taken into account.

Case II.—G. W. S., aged thirty-six years; married; an employé of the fire department; American.

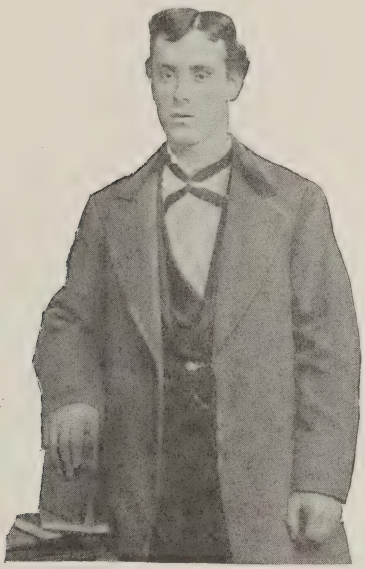
Family history: This is practically negative. His father was killed by an accident when forty years of age. His mother is living and well. Has one brother and one sister, both of whom are also well. All of his people, he states, were of average build, though his mother and an aunt on his mother's side incline to obesity. He has a son of thirteen who is in every way normal.

Personal history: He had none of the illnesses of childhood, but when two and a half years old lost the sight of his left eye by a fall from a sled. His

mother states that he had a convulsion as a result of this fall, and that he was ill for some weeks. However, he made a good recovery, and has had no illness of moment since that time.

According to a photograph taken when the patient was twenty-one years of age (see Fig. 3), he seems to have been a tall young man with perhaps ordinary features. The chin, however, seems already a trifle pronounced. The hands, too, though large, are not markedly so, while the fingers are long and even slender. The patient himself thinks that it was at this age

Fig. 3.



G. W. S., aged twenty-one years.

or shortly after that he first noticed that his hands were getting bigger. Later on he began to get larger in face, hands, feet, and body—he seemed, as he expressed it, to get bigger all over. The process must, however, have been very gradual, for a photograph taken when he was twenty-eight shows less change than might have been expected. However, his nose and chin had already become large and pronounced. The cheek bones, too, from having been rather flat, had become more prominent. The patient stated that he had increased steadily in weight, and that when thirty years of age he weighed two hundred pounds.

No alcoholic or venereal history.

Present condition: His features are so very large as to attract immediate attention, while his hands and feet are truly enormous. On stripping him

we find that this enlargement is not by any means limited to these parts, but is shared by the body as a whole.

Studying his face (see Fig. 4), we find that his supra-orbital regions are occupied by huge bony prominences; that the cheek bones are so excessively hypertrophied as to give a knobbed and angular appearance to the cheeks; that the lower jaw is positively massive; and that the nose, though

FIG. 4.

G. W. S., aged thirty-six years.

well shaped, is very large. Due to the elongation of the face and the excessive prominence of the malar bones, deep depressions have formed to either side of the nose, and these, together with the distorted features, give the countenance an almost hideous aspect.

The tongue is much hypertrophied. The lower jaw projects far beyond the upper, so that the teeth do not meet. Some of the molars have been lost.

The ears also are very large.

On looking at the back of the head we find that the external occipital protuberance, the inion, has become so hypertrophied that it forms a massive conical projection more than an inch above the general surface of the skull. The face and mucous membranes are perhaps a trifle pale, but less so than in Case I.

Turning our attention now to the hands, we find that they are excessively broad, thick, and spade-like, while the fingers seem rounded and stumpy. As in Case I., the normal transverse furrows are much exaggerated. The nails also are broad, flat, and striated. The feet closely resemble the hands in their essential features.

Unlike Case I., the hypertrophy is not limited to the extremities, but affects apparently the limbs and trunk in their entirety. A glance at Fig. 4 reveals undoubted hypertrophy of the arms and chest, and this is also true of the lower portion of the trunk and legs. The increase in size appears to affect bone and muscle alike. The hypertrophy of the muscles is, indeed, very striking. The muscles, however, seem soft rather than firm, and although the patient asserts that he is very strong, the dynamometer registers but 72 in the right hand and 74 in the left.

The brother of this patient is an unusually small-featured and small-limbed man, so that the contrast between them is very striking.

The following are the measurements that were taken:

	Right. Inches.	Left. Inches.
Length of hand	8¼	8¼
Circumference of hand at knuckles	10¼	10½
" " metacarpus with thumb	13	13
" " forefinger, proximal phalanx	4	4
" " wrist	8¾	8¾
" " forearm, junction of upper and middle third	13⅜	13½
" " arm	15	15⅛
Length of foot, heel to tip of great toe	11	10¾
Circumference of foot at ball of great toe	11¼	11⅜
" " " " instep	12¼	12½
" " " across instep and heel	15	15
" " ankle	10	10
" " leg at junction of upper and middle third	17¼	17⅛
" " thigh at junction of upper and middle third	23	23⅛
Length of nose (root to tip)		2¾
From zygoma to zygoma		5½
Depth of chin		2½
Circumference of neck		17
Distance between acromions measured over back		17
Circumference of chest, at nipples, expiration		44
" " " " " inspiration		47½
" " " xyphoid cartilage, expiration		43½
" " " " " " inspiration		47½
" " abdomen		41½

A comparison of these figures with those of Case I. reveals a decided difference as regards the forearms and legs. These are much larger in Case II. As regards the foot, the same holds good as in Case I. There is a similar formation of tissue at the back of the calcaneum and the same appearance of a negro's heel. It is, however, less pronounced than in Case I.

There is present here no sense of general weakness. Indeed, the patient, deceived by his hypertrophied muscles, believes himself to be very strong. As in Case I., there is no alteration in cutaneous sensibility. The knee-jerks are decidedly minus.

An examination of the eyes by Dr. de Schweinitz revealed the left eye to be sightless, vision having been destroyed by ophthalmia in childhood. The field of vision of the right eye, both for form and color, is exactly normal, presenting the full physiological limits. With the exception of some anæmia of the nerve-head, which is probably dependent upon some general anæmia, the examination of the right eye is entirely negative.

The other special senses likewise present nothing abnormal.

The urine has a specific gravity of 1020, and, as in Case I., is negative to all tests.

The patient never suffers from headache. Occasionally, however, has some tinnitus, but which is present only at times. No mental depression or other mental symptoms. Sleeps, however, a great deal. Is very often drowsy during the day. Sweats excessively, especially about the head. Appetite and thirst both very great. Is sexually inactive; not living with his wife.

Weight at present two hundred and fifty-five pounds. Very little of this weight is made up of fatty tissue.

There is no kyphosis.

The above cases are interesting because of the manner in which they were found, and because of the absence of headache and of decided eye-symptoms. Regarding headache, upon which Souza-Leite lays so much stress, I find it mentioned in only a little over one-third of the recorded cases. The general invasion of the limbs and trunk by the hypertrophy in Case II. must also be looked upon as significant.

In the study of any obscure disease there is always danger of ascribing to facts which bear the relation of mere concomitance, a relation of cause and effect. Pierre Marie, to whom belongs the great credit of having isolated acromegaly, and of having defined its position as a clinical entity, has advanced the view that the affection is due to disease of the pituitary body, just as myxœdema is due to disease of the thyroid gland. It is true that he has not presented this view as though abso-

lutely established by the evidence, but rather as a very probable hypothesis. I believe, however, that the facts at hand are more in favor of its rejection than of its acceptance. It is undoubtedly true that in the larger number of autopsies thus far held the pituitary body has been found enlarged, but if such enlargement really be the cause of acromegaly, it should be found in every instance—it should be universal. In a number of cases, however, it has not been recorded, and in several in which it has been carefully looked for, notably the case of Virchow,[1] the pituitary body has been found absolutely normal. Again, observations are not wanting to prove that the pituitary body may be enlarged without the concomitance of acromegaly, as, for instance, in the case recently reported by Packard.[2] Further, it appears that the pituitary body may be absolutely destroyed[3] and yet no symptoms of acromegaly appear. Weir Mitchell some years since reported a very remarkable case (in which I myself studied the specimens) in which an aneurism had formed in an anomalous branch of the circle of Willis. In addition to destroying the optic chiasm it had hollowed out the entire cavity of the sella Turcica, eroding its surface and boundaries. From the notes of the autopsy the aneurism occupied the cavity of the sella Turcica to the exclusion of everything else. Here, then, we have an instance in which Nature has performed a vivisection experiment, has destroyed the pituitary body without producing the symptoms of acromegaly, for had they been present it is safe to assume, from the reputation of the observer, that they would have been recorded.

When we consider the clinical records and autopsies of acromegaly still further, we find that there is hardly a gland in the body which has not at some time or other been reported as

[1] Berliner klin. Wochenschrift, 1889, xxvi. p. 81.

[2] F. A. Packard: The American Journal of the Medical Sciences, June, 1892, p. 657, third case.

[3] S. Weir Mitchell: Journal of Nervous and Mental Diseases, January, 1889. "Aneurism of an Anomalous Artery, causing Antero-posterior Division of the Chiasm of the Optic Nerves and producing Bi-temporal Hemianopsia."

enlarged: the pituitary body, the thyroid gland, the thymus, the lymphatic glands, the spleen, the kidneys, the lobes of the liver, and the testicles. Certainly with these facts before us we should hesitate to ascribe the affection to disease of any one of them, even if, as in the case of the pituitary body, its hypertrophy is quite frequent. It would be safer to ascribe to the hypertrophy of this body merely the same value as that which must be assigned to the hypertrophy of the other glands, namely, a participation in some obscure and general morbid process. Finally, when we recall such post-mortem findings as hypertrophy of the sympathetic system and peri- and endoneuritis, a legitimate doubt is raised as to whether the glandular apparatus plays any active part whatever.

DISCUSSION.

Dr. Charles Shaffner: I have seen a case where the hypertrophy involved the right upper extremity. The arm was uniformly enlarged, and both the bones and the muscles seemed to be involved. When I first saw the patient she was fourteen years of age, and came on account of interstitial keratitis, the result of hereditary syphilis. Anti-syphilitic treatment had no effect upon the enlargement, although it improved the keratitis very much.

Dr. F. A. Packard: I would like to ask Dr. Dercum in regard to the presence or absence of vascular changes in his two cases. In most of the cases hitherto reported there has been a condition of varicosity of the veins throughout most of the body. In the patient that I showed to the College early in the year, the veins are all enlarged. In the lower extremities there were marked varicosities, and the patient had twice been operated on for hemorrhoids.

Dr. Dercum: The veins in these cases are enlarged and varicose in the lower extremities.

Both of these patients suffer from drowsiness at times. They have both made this statement unsolicited. They sometimes go to sleep while talking. Dr. Packard's case would sometimes go to sleep while eating.

UTERINE FIBROMATA; REMOVAL OF TWENTY-SEVEN, WITH TWO DEATHS.

By J. M. BALDY, M.D.

[Read November 2, 1892.]

It is not many years since it was almost universally taught that fibroid tumors of the womb were harmless growths, and that their presence might with impunity be ignored. Since that time there must have been a change either in women or in the character of these neoplasms. Possible it is that the profession, being only too fully awake to the dangers of the one rational method of treatment, preferred excessive caution to legitimate boldness. However this may be, the great advances in abdominal surgery have put this disease, together with many others, within the category of affections that can be cured with comparative ease.

The symptoms to which the affection gives rise are variable, and are dependent to a great extent, with possibly a single exception, upon the size and shape which the growth may assume. The fibroid tumor in itself, so long as it remains a true fibroma, will occasion little trouble other than the induction of uterine hemorrhage. As it grows its presence will sooner or later induce irritation of the peritoneal surfaces, thus giving rise to considerable pain, and oftentimes distention. The irritation may even amount to an inflammation, which is apt to be kept up or relighted, frequently or not, according to the amount of exposure to determining causes.

Pressure, within the pelvis especially, gives rise to many distressing symptoms, not limited to the pain which it causes. The bladder is most frequently irritable and incapacitated for holding the urine for any great length of time; it has been my experience to find this organ many times in a condition of chronic inflammation, the urine being loaded with pus. The bowels are usually constipated, due to a great extent to the mechanical obstruction caused by the pressure; it is a matter of surprise to me at times, when handling these tumors, how any fecal matter whatever has been able to pass the pelvic brim. Patients carrying a tumor of this class are liable to repeated attacks of peritonitis, attacks which are not always sufficiently violent to endanger life materially, but which render the woman an invalid for weeks, and often months. It would seem at first blush that the tumor of a woman who had suffered from one or more attacks of peritoneal inflammation must of necessity be adherent to surrounding parts. This is not the case, however, excepting where the inflammation is septic or specific. A septic or specific inflammation of the serous membrane almost invariably leaves adhesions in its train; a simple traumatic peritonitis is likely to clear up without such serious consequences. Although fibroid tumors are likely oftentimes to be complicated by diseased uterine appendages, and are thus open to the chances of an adhesive inflammation, yet it is a fact that the majority, even of the complicated ones, are accompanied by few, if any, adhesions, even after repeated inflammatory attacks. The amount of suffering attendant upon these growths is, as a rule, in direct ratio to the complications, and has little or nothing to do with the size, unless that has become enormous. The sufferings of a woman carrying such a growth as large as a full-term gravid womb are frequently not nearly so great as those of one with a tumor which barely rises out of the pelvis. Much has been written concerning the organic changes in various organs caused by the pressure of fibroid tumors. These changes are as often mythical as not. It has occurred to me to find chronic cystitis and, more frequently, a thickened parietal peritoneum,

but otherwise in the twenty-seven uterine fibroids which I have removed the other organs of the patients have been healthy. In two or three of them I have found albumin in the urine; but this was evidently due to pressure, as, except in one case, it cleared up almost immediately after the operation. In all of my patients, with this one exception, the kidneys have been healthy; the ureters have never been observed to be in an abnormal condition; the bowels have been normal, and the other abdominal organs have remained unaffected.

I find myself, therefore, unable to suscribe to the oft-repeated statement of the dangers of irreparable organic changes in the abdominal organs due to the presence of fibroid tumors of the womb. It has been my lot, however, frequently to find sarcomatous changes taking place in the tumors themselves—tumors which had formerly been apparently perfectly healthy. Diseased uterine appendages accompanying the growth have been observed in the majority of cases coming under my notice; pus tubes, hydrosalpinx, old chronic salpingitis, with great hypertrophic enlargement of the ovaries, ovarian cysts, and ovarian abscesses have been the most frequent complications. One case of tumor of the broad ligament, or, more correctly, uterine tumor, which had grown into and between the folds of the broad ligament, was complicated by adhesions to several coils of intestines; these being released and the tumor being left *in situ*, on account of the risks of the operation, the symptoms all subsided, and there has not been a return of any one of them, now more than a year and a half since the operation.

The old methods of treating uterine fibromata have not much to recommend them. In many cases they will probably answer as well as anything, because many cases require no treatment at all—in other words, the mere presence of a fibroid tumor is no warrant for dosing the patient with purgatives, ammonia, ergot, hydrastis, electricity, and similar remedies. It must not be expected that the tumor can be gotten rid of short of surgical methods, and these are to be reserved for special cases. Where it comes to dealing with symptoms, however, these remedies all have their place, and are capable

of doing much good, provided they are used judiciously. Rest in bed, purgatives, counter-irritation, hot douches, electricity, etc., will frequently relieve the pain, which is most often due to peritoneal irritation and which should be treated on general principles. The curette, especially in small growths, ergot, hydrastis, atropine, electricity, and similar remedies come into play for the hemorrhages, and will temporarily control the bleeding. If one is able to accomplish this much, control of pain and hemorrhage, he has often accomplished all that the patients desire, and it would be hard to persuade them into having an operation performed. If the inflammatory attacks are not recurring and the hemorrhage is not excessive, provided the tumor is not so large as to cause trouble by its size, the patient may rest content with her present condition, especially if she be approaching the menopause. It by no means follows, as has so often been taught, that most of these neoplasms cease to grow at this period of life.

On several occasions has it been found necessary in my experience to remove the uterus, together with the growth, in women forty-eight or fifty years of age. Yet an occasional case occurs in which the change of life ends the period of suffering, and the growth ceases to cause active symptoms. In view of this fact, if the woman is near the time of her change, it may be worth her while to wait, provided her symptoms are not too distressing. Each of these cases must be a law unto itself as far as the consideration of surgery is concerned, and yet fairly accurate lines can be laid down along which it will be safe to advise any given patient.

1. All rapidly growing fibroid tumors in young women (before thirty-five) should be removed; and many of the same kind as late as forty years of age. Removal is likewise necessary in:

2. All cases in women under forty, where there is such loss of blood as to enfeeble the general health, and which is not readily controlled by treatment;

3. All cases under forty in which there are frequent recurring attacks of peritonitis;

4. Cases which have gone several years past the menopause, with excessive uncontrollable bleeding or recurrent attacks of peritonitis.

Such advice as this would a few years ago have seemed unwise and unsafe; but, in view of our present successes in the surgery of such growths, we are justified in adopting more radical measures than formerly. No one can doubt that a woman is better and safer without a fibroid tumor than with one, and, therefore, the only questions worth considering are with how much safety can they be removed, and is there any surer or safer method of removing them than by extirpation?

Oöphorectomy has been proposed and practised in times past for the purpose of accomplishing this object, but has proved itself both unsatisfactory and unreliable, besides being almost as dangerous as the removal of the tumor. The operation, excepting for small pelvic tumors, has passed out of use. The claims made for electricity, so far as the removal of fibroid tumors is concerned, have proved, as predicted, fallacious, and to-day none but the most enthusiastic electro-gynecologists are making any claims in that direction, and these claims are made invariably without proof. The removal of a healthy, non-adherent fibroma of the womb should be and is attended with but a small percentage of danger. Of the twenty-seven tumors of this character which it has been my fortune to remove by the extra-peritoneal method, death occurred in but two cases (about 7 per cent.). In neither of these cases could the fault be placed on the operation, but rather on the operator and the previous treatment. The first death was that of a colored woman with a tumor which weighed much over twenty pounds. During the operation a towel (which had not been prepared for the purpose) was placed in the abdominal cavity to hold the intestines in place while the necessary steps for treating the pedicle were carried out. This towel infected the peritoneal cavity, and the woman died five days later of purulent peritonitis. The second patient was a white woman whom I had seen three years before in good health, with a healthy, movable tumor. At the time of the operation she was an emaciated, bedridden

woman, with a rapid, feeble pulse, a high temperature, and deeply septic. The tumor was fixed by adhesions, and a sinus which discharged pus freely was found on the outside of the left labium majus, and pus was pouring from the vagina. The intestines and omentum proved to be adherent to the tumor, the tumor was adherent to the whole pelvis, both ovaries contained pus, and the external sinus opened into the left ovary, and both ovaries and tubes were universally adherent. When the uterus, tumor, Fallopian tubes, and ovaries were all freed, delivered, and removed, there remained sinuses into the vagina and into the cellular tissue about the vaginal sheath. These complications all followed a course of treatment by electropuncture in the hands of an expert. That the patient died of purulent peritonitis was surely no discredit to hysterectomy. With these two exceptions, all patients from whom I have removed tumors of this character by the method which I almost invariably adopt, have had as easy a convalescence as is the case with patients who have had an operation for a simple ovarian cyst. The method used has been that known as the extra-peritoneal treatment of the stump.

In this operation the tumor is turned out upon the abdomen, and a wire clamp is placed about the base of the neoplasm, in order that it may constrict it and control the bleeding. A transfixion pin is placed above the wire to keep the clamp from slipping up or the stump from being pulled down through its grasp. The stump is then fixed in the lower angle of the incision and the abdominal wound closed. Whenever there is any inclination for the stump to bleed, the wire is tightened, and all danger of hemorrhage is thus avoided. The advantages of this plan of treatment are, first, that the stump is under perfect control, and if it shows any signs of bleeding the leakage can be checked instantly by tightening the wire; secondly, if there be any septic matter in the cervical canal or about the stump, it is outside the abdominal cavity, and may suppurate with safety; thirdly, all raw or denuded surfaces are outside the peritoneal cavity, and there is no chance for adhesions to form between them and the intestines.

The disadvantages urged against this method of treating the stump are that the free action of the bladder is interrupted and that hernias are likely to follow at the point where the stump was originally fixed. The bladder certainly is irritable so long as the wire is kept on the stump, but this is corrected as soon as the stump has sloughed away or the wire is taken off. In but two cases have I seen hernia. In the one patient it appeared at the site of a drainage-tube, and being more than an inch above the pedicle, could consequently not be put down to anything but drainage. The second hernia appeared at the site of the old stump. The operation had been done in the case of a large sarcomatous uterus, which was cut away and the stump treated outside the peritoneal cavity. As a matter of course, there was not good union between the sarcomatous tissue of the stump and the surrounding tissues, and I was not at all surprised when the woman returned with a hernia. That hernia did not appear in any of the other cases is pretty fair proof of the fallacy of this criticism. The danger of septic peritonitis from a sloughing stump is a familiar objection from those who are opposed to this method of treatment. Such an objection must be born of lack of experience. In no single case was there any such accident, although many of the stumps sloughed; in fact, it is this very danger of septic peritonitis that is avoided by the extra-peritoneal method of treatment of the stump.

Occasionally a fibroid tumor of the uterus cannot be brought out of the abdominal wound so as to place a wire about its base. This is caused by the fact that the growth has split open one or both broad ligaments and has developed between its folds. In these cases it becomes necessary, if the tumor is to be removed, to tie off the broad ligaments and secure both the ovarian and uterine arteries; in which case it is probably best to then drop the stump back into the abdominal cavity or remove it entirely. However advantageous and easy this method may be in skilled hands, experience has taught us that, as yet, the extra-peritoneal method has proven the safest and surest, and the time has

not yet come for substituting for it other methods which have not been fully tried and matured. For the present, at least, a beginner in hysterectomy should think of but one method of treating the stump—the extra-peritoneal—where that method is practicable; and, as a matter of fact, in the vast majority of cases it is practicable. It is both the easiest and safest method to begin with, and if after mature experience one wishes to deviate and adopt a method that will give a less prolonged convalescence, he may find, if he have skilled fingers and a cool head, some of the intra-peritoneal methods, now being rapidly developed, preferable in some respects but much harder of performance and with many more elements of danger.

DISCUSSION.

Dr. Charles P. Noble: I find myself closely in accord with Dr. Baldy with reference to this subject. Like him and like most others, I have passed through the stage of looking upon fibroid tumors as tumors which should not be operated upon. Why should a woman be an invalid for ten or twenty years when she can be restored to health by an operation which, if done early, before complications arise, is not dangerous? When I began practice it was not customary to operate on fibroid tumors. We then had an opportunity of studying the natural history as modified by the medical treatment in vogue. Such treatment acts simply as a palliative, and nothing more.

There are several points that impress me. First, with reference to attacks of peritonitis as a complication. I believe that what is assumed to be peritonitis is very often not so; that the inflammatory trouble is in the tumor itself. I have operated in cases of fibroids in which there were supposed to have been well-marked attacks of peritonitis, and found no adhesions. Dr. Baldy explains this by saying that the peritonitis was non-septic. I would explain it by saying that there had been a mistake in diagnosis. Undoubtedly peritonitis does often complicate fibroid tumors, and in these cases it originates from disease of the appendages, as Dr. Baldy has very properly stated. I believe that the pain which often accompanies fibroid tumors of moderate size is due not so much to pressure as to inflammation in the ovaries and tubes.

The old theory in regard to the influence of the menopause upon fibroid tumors will certainly have to be modified. There is no doubt that there is an

element of truth in this theory, as there is in most theories that have obtained almost universal acceptance. It is also true that change of life often does not cure fibroid tumors, even symptomatically. I have removed one tumor that did not become apparent until after the woman was fifty-six years of age and had passed the menopause eight years. It had attained the size of a six months' pregnancy in a short time. That is not a single experience. In soft myomata it is quite common for the tumor to grow more rapidly after the menopause than before. I think that the class of tumors most modified by the menopause is the hard nodular tumor, or cases where there are several hard tumors of the uterus.

I have myself done the operation of removal of the uterine appendages for fibroid tumors about a dozen times, and in my hands the results have been very good; in fact, they were all that could be desired. I did not expect the tumors to disappear, and, as far as I know, they have not disappeared; but in the cases that I have been able to follow, the tumors have diminished in size and the hemorrhage and other symptoms have been relieved. I have therefore no complaint to make of this operation in small hard tumors. The experience of all surgeons is that in soft fibroids this operation exerts no influence, and in very large tumors the results have not been satisfactory.

I am heartily in accord with Dr. Baldy in reference to the stump. Undoubtedly the best results have been gotten by the extra-peritoneal treatment of the stump, and the only reason why operators are endeavoring to perfect an intra-peritoneal treatment is the long time required by the former method, it being six or eight weeks before the wound is healed, and this is undoubtedly an objection.

It is said by every author that hernia is common after this operation. I must confess that in my own experience I have not met with hernia after the removal of fibroid tumors, nor have I seen it in the practice of others, but I have not seen a large number of cases. I suppose, however, that there must be a basis for this universal assertion, and that Dr. Baldy's experience must be exceptional.

Dr. John C. Da Costa: I congratulate Dr. Baldy upon his results, but must say that I am a little more conservative, and in selecting cases for operation think that it is safer to draw the lines a little closer. I would advise operations in rapidly growing painful fibroid tumors without reference to the age of the patient, and do this because I believe that rapidly growing painful fibroid tumors are those most likely to degenerate into malignant tumors. As an instance of this, Dr. Baldy will remember a case of this kind that he saw with me, in which, after opening the abdomen and shelling out an apparent fibroid as large as two fists, I shelled out another as large as an orange. The first one was cut open and proved to be a fibro-sarcoma, while the other was a pure fibroid. Hysterectomy was then done, and the uterus found to be a mass of sarcoma.

In regard to the size of the tumor: Unless the size proves a great inconvenience, it is better to let the tumor alone except when there are other symptoms. This is illustrated by another case which Dr. Baldy also saw. The woman was near the menopause and had a large fibroid, which had not grown for five or six months. Under medical treatment the symptoms practically disappeared. The patient left the hospital much improved, and finally drifted into another surgeon's hands (not Dr. Baldy's). She was operated on and died promptly within twenty-four hours. That shows the advantage of letting such cases alone.

I am rather of the opinion that in extra-peritoneal hysterectomy there is more risk of hernia, but the safety of this method outweighs the risk of the hernia. Hernia can often be avoided by care in doing the operation, by bringing the pedicle close to the bottom of the wound and the peritoneum closely around it so as to confine the stump of the uterus very closely; then closing the cut above it so as to secure firm union.

Dr. Baldy: In regard to hernia, I would say that, as we all know, in medicine we are apt to have statements handed down through a long series of books, which on careful investigation proved to be fallacies; I believe that this is largely the case in reference to the assertion of the frequency with which hernia occurs after hysterectomy. The fact that so many authors speak of hernia is simply a proof that they have accepted what someone before them has said, and repeated it without any practical experience in the matter. The question whether or not there will be hernia depends largely upon the treatment. When a large stump was left, the risks of hernia were much greater. Now the stump can be brought down to a very small size. I keep patients in bed for six or eight weeks, and I think that practically that is one of the secrets of success in avoiding hernia. As I have said, I have had two hernias follow this operation. One was due to the non-union of sarcomatous tissue to healthy tissue, and the other did not occur at the seat of the stump, but at the drainage opening. In dispensary practice I see many patients on whom abdominal section has been performed by various operators, and I see many hernias that have followed cœliotomy, but I have yet to see a hernia that has followed hysterectomy.

I am in accord with Dr. Noble in regard to peritonitis. I do not believe that one-third of the cases of so-called peritonitis in fibroid tumors are really attacks of peritonitis. In the majority of cases it is mere irritation. I have operated in cases presenting at the time the symptoms of a well-marked attack of peritonitis, and have failed to find evidence of inflammation in the peritoneum. I believe also that the pain is often resident in the appendages.

A CASE OF CONGENITAL DEFICIENCY OF THE LOWER EXTREMITIES.

By GEORGE ERETY SHOEMAKER, M.D.

[Read November 2, 1892.]

The child before us was born and now lives in Maine. She was recently sent to this city, and admitted to my wards at the Methodist Hospital, for the purpose of securing mechanical support. She is shown here simply as a case of congenital deformity, though I may say that she has learned since coming here to walk readily by the aid of crutches, upon rigid rods attached to a plaster-of-Paris cup enveloping the pelvis, the weight of the body resting entirely upon the tuberosities of the ischia, as in the normal sitting posture.

The permanent apparatus (shown in Fig. 1) is simply an elaboration of the same idea—a pelvic cup with two rigid metallic limbs fastened together and jointed at the sides of the pelvis. There is at this point a simple lock, which can be loosened when the patient wishes to sit down. The part which surrounds the pelvis is made of rawhide moulded over a plaster cast in the same way that an ordinary leather spinal jacket is made by Mr. Gemrig, who has constructed the apparatus here shown.

Owing to the activity and early age of the case, it is not considered advisable to complicate the apparatus by knee-joints, though, were she an adult, a fairly useful joint might be devised for that situation.

The child is nine years of age, and is one of seven living

children. Her parents are living but present no deformity; nor are the other children deformed.

From the standpoint of teratology the case is of much interest. Deformity of the lower limbs alone is said by Geoffroy St. Hilaire to be more rare than that of the upper, and also more rare than a deficiency in all four. In Ahlfeld's work on *Deformities* I was unable to find a case of precisely the same type. The recent work of Dr. Hirst and Dr. Piersol does not do more than refer to a classification in the folios so far issued. However, M. Breschet (*Bulletin de la Faculté de Médecine*, t. iv., p. 328) describes deformities in an adult male curiously similar to the present. The man was at that time (early in the present century) a great travelling curiosity. In his case and in this, the rudiments are very unlike on the two sides, but, curiously enough, the conditions present are exactly reversed so far as the sides are concerned.

One may recall the general division of members into four segments which is observed in man and many higher vertebrates: first, the one nearest the body for the attachment of the member to the trunk, illustrated in man by the shoulder-girdle, or half the pelvis; second and third, the intermediate segments, as the arm and forearm, or the thigh and leg; finally, the end segment, represented by the hand or foot.

In the case before us the first or pelvic segment remains apparently normal in bony development, with no evidence of abnormal origin of muscles. For instance, the glutei are well developed, as are apparently the psoas and iliacus, and the upper ends of muscles arising from the front of the ilium and which would flex a thigh.

However, the insertions of these muscles into the rudiments of the extremities are obscure; the bony elements of the rudiments are so small and so abnormal in shape that in the living subject no certainty attaches to any attempt at identification. The difficulty is at once understood when it is stated that both rudiments are buried to a considerable degree within the contour of the buttock. No hip-joint is present on either

side, and probably no femur, though the structure of the left rudiment is less clear than that of the right.

In the right rudiment the soft parts resemble to some extent a small leg and foot, but the resemblance is entirely superficial. Instead of the normal thirty bones, the limb below the pelvis

FIG. 1.

Permanent apparatus applied.

appears to contain only two bones. There is no hip-joint and no femur. There is one long bone, probably tibia, which has a musculo-ligamentary attachment to the pelvis in front of and below the anterior superior spinous process of the ilium, where

it is buried beneath the integument and tissues of the trunk. This bone is about five inches in length, and extends downward and forward to the very tip of the single toe-like extremity.

Fig. 2.

Sitting position.

There is no nail upon the latter, but the skin is hardened by use. The other bone is short and irregular, not over an inch in length, and is felt behind and a little above the lower end of the longer bone. It is freely movable, gives substance to the cushion used as a heel, and is very probably a rudimentary fibula.

The tarsus, metatarsus, and phalanges are completely absent. In other words, the only portion of the limb present on this side is an imperfect lower intermediary segment. There is no true joint present, and the free voluntary movements in all directions take place through the muscular attachments of the

FIG. 3.

Position assumed when walking on the hands.

single long bone to the pelvis. The limb may be elongated about two inches by traction downward, the upper end of the long bone sliding along the pelvis. It still has a large degree of strength, and the girl is able to stand upon the two rudiments, when the buttocks are raised so as to clear the floor about half an inch.

The left extremity is much weaker and more limited in motion than the right, though it contains more parts. The foot segment is represented by a single well-formed toe with its metatarsal bone. A toe-nail of ordinary appearance is observed. There is no tarsus. A rudimentary tibia and fibula about four and three-quarters inches long, and very thin and weak, may be traced partly within the buttock. From the upper end of the tibia a stronger mass extends backward toward the acetabulum, but its identity as a femur is uncertain, because of its being buried in the buttock. The whole limb is flexed or folded upon itself, till, in standing, the finger-like foot lies upon the floor, and the fibula and tibia lie almost horizontally upon it, and are in contact with it for a considerable distance; this throws what should be the knee in front of and below the anterior superior process of the ilium, but it is completely covered by the tissues of the anterior part of the buttock or trunk.

The limb cannot be straightened to any considerable degree, and its chief motion is backward, though that is quite limited. There are possibly present some elements of every segment of a normal limb, though no positive evidence of a femur exists.

There is no deformity in any other part of the body. The large development of the shoulders and upper arms is due to over-use. The elbow-joint over-extends about an inch and a half beyond a straight line, as though from its unwonted strain. The triceps on each side is better developed than the biceps, because she uses her arms as legs in walking. The biceps is assisted in maintaining rigid extension by the chocking of the elbow-joint. The triceps is not so assisted, and does more work. The genitalia and anus appear normal, though the labia majora are rather small. It is usual for deformed individuals of the kind to be sexually deficient, though the man to whom allusion was made as resembling this case was the father of several children.

The child is strong and active, and has always been well. The height when sitting is 27 inches, which is increased to 29 inches when she "stands" resting on the rudiments and bal-

ancing with hands upon the floor. In this position the weight is borne on the last phalanges of the thumbs and on the meta-carpo-phalangeal joints. The chest at the nipple line measures $23\frac{1}{2}$ inches after expiration, and $25\frac{1}{2}$ inches after inspiration; waist at umbilicus, $20\frac{1}{2}$ inches; circumference of hips, $24\frac{1}{2}$ inches. The general development corresponds to that of the average girl at her age.

She walks at times by using her arms like crutches, and swinging the body forward between them. She also walks on the stumps, or, more properly, on the buttocks, swinging first one and then the other forward. She walks readily upon her hands with the pelvis high in air, as shown in the photograph. Her intelligence is good, though she has been much repressed, her family being ignorant; she has no education, though that is not for want of capacity.

No perfectly satisfactory explanation can be given for the failure in development. It may simply be said that any theory of intra-uterine amputation or constriction is to my mind disproved, at least on the left side, by the persistence of the single well-formed toe and its corresponding metatarsal bone, with the complete absence of traces of the other toes. Abnormal distribution of blood-supply could not account for this; and we are thrown back upon trophic influences, probably of central origin, whose nature in the last analysis is unknown.

It may be recalled that there is a classification of monstrosities which depends upon the development of the limbs. Thus a monster without limbs belongs to the class *Amelus* (μέλος, a limb). One with two limbs fused together becomes *Symelus*. The *Phocemelus* has a terminal segment of a limb, for instance the hand, attached directly to the shoulder-girdle without the intervention of the intermediary segments, as the arm and forearm. This arrangement is normal in some marine animals, hence the name Phocemelus (φώκη, a seal, μέλος, limb).

In the *Hemimelus* two distal segments are wanting. It will readily be seen that the present case belongs to none of these groups, but must be classed as *Ectromelus* (ἔκτρωσις, *abortion*),

according to the classification of Geoffroy St. Hilaire and others, there being simply a failure in development.

The accompanying illustrations are from photographs.

DISCUSSION.

DR. FRANK WOODBURY: I recall the case of a man about twenty-six years of age, with a similar deformity, who gave public gymnastic exhibitions. He became quite expert in the use of the arms as organs of locomotion, and was able even to jump from considerable elevations. He seemed to enjoy perfect health, and, as in this case, there were no other evidences of deformity or malformation.

With regard to the intra-uterine cause of such a condition several speculations might be indulged in, one being injury to the lumbar enlargement of tbe cord affecting the development of the lower extremities at a very early period of growth; another, malformation of the arteries, either the common iliacs or their continuations being smaller in size than normal, and carrying a deficient quantity of blood to the lower extremities, which, consequently, must suffer in nutrition. Where the deficiency was due to some central nerve-disorder, we should expect atrophy of the muscles arising from the pelvis as well as of those further down, and we should also expect all the parts of the lower extremity to be represented in the part, although in a rudimentary form. Where there was a defect of the bloodvessels, cutting off the supply of blood, we should still expect the foot and other parts to be present, but in a miniature form, unless the blood-supply were suddenly shut off, in which case sloughing might result.

A number of children are born with knots in the umbilical cord, showing that the entire body must have slipped through a coil of the cord. Sometimes the effects are fatal to the fœtus. It has been suggested that the lower extremities might be entangled in the cord in such a manner that constriction might be produced, and thus interfere with their development. I am aware, however, that this cause has been considered very problematical, if not impossible.

The most plausible explanation is traumatism—that is, fracture of the femora, possibly by over-extension of the thighs, or by the muscular contraction of the uterus under some strong influence. This occurring at an early stage might be followed by arrest of growth, and perhaps entire loss by something approaching gangrene. This would account for the absence of rudimentary feet. Of course, these are simply speculations as to the cause of this malformation, the true explanation of which is at present unknown.

A CLINICAL STUDY OF ELEVEN CASES OF ASIATIC CHOLERA TREATED BY HYPODERMOCLYSIS AND ENTEROCLYSIS.

By JUDSON DALAND, M.D.

[Read November 2, 1892.]

This evening I shall give a short description of the clinical history and treatment of eleven cases of Asiatic cholera that I had the opportunity of observing at the Swinburne Island Hospital in New York Bay last September.

The *first* case represents a group that may be called mild cases, and occurred in a Russian, aged sixteen, admitted to the ward Sept. 9th from the steamship Bohemia. He complained of abdominal pain of moderate severity. His temperature was 99.2° F., pulse 94, and respirations 24. Three more observations, which were continued until midnight, gave the same results. It was worthy of note that at no time was the temperature below normal. The first act of vomiting after admission brought up a greenish liquid mixed with mucus. The diarrhœal discharges were whitish in color and rice-water-like in character. The spirillum of cholera was detected in the discharges. The pulse was of fair volume. The patient slept well and passed a normal quantity of urine. After remaining in the cholera ward three days he was discharged fully convalescent.

His treatment was as follows: Upon admission he was given ten grains of calomel, and afterward he received one grain every hour for six doses. He was also given two quarts of a 1 per cent. solution of tannic acid at a temperature of 104° F., by enema, every three hours. At no time was it necessary to resort to subcutaneous injections of the $\frac{6}{10}$ per cent. solution of sodium chloride. He was also given twenty minims of whiskey hypodermatically, and occasionally one-eighth grain of morphine.

The *second* case was a typical example of an ordinary attack of Asiatic cholera, in a Russian, aged seven years, admitted from the steamship Bohemia, September 26th, at 8 P.M., with a temperature of 96.2° F., pulse

106, respirations 22, and the surface of the body extremely cold and cyanotic. The face was pinched, especially in the nasal region; it was markedly cyanotic, and the eyes were deeply sunken and surrounded by dark circles. The pulse was feeble and small, and was counted with difficulty. The patient at once received a warm plunge bath, and her stomach was then washed out with one pint of a 1 per cent. solution of tannic acid at the temperature of the body. Shortly afterward she vomited twenty-three ounces of fluid. She remained in about the same condition during the night, but the passage of a moderate amount of urine encouraged us to hope for a favorable issue. On September 27th her pulse was 104, her temperature 97.4° F., and her respirations 18 to the minute. The cyanosis persisted. There was no vomiting, but she had frequent copious discharges from the bowels, composed chiefly of the tannic acid solution which had been injected *per anum*. Later in the day improvement began, the temperature varied between 100.2° and 100.4° F., a large semi-solid stool was passed; the patient slept moderately well during the night, but complained greatly of thirst. A sufficient quantity of urine was excreted, and the liquids from the bowels were chiefly composed of tannic acid solution that had been given by injection. She became very restless, but the following day was much brighter and began to notice her surroundings. Her dejecta changed in character and became greenish in color and somewhat frothy. On September 29th she slept well and her bowels were moved only after giving the intestinal injection of tannic acid solution. In the afternoon her improvement was so marked that it was decided to suspend these injections. Her temperature varied from 97.8° to 99.4°, the pulse from 96 to 120, and the respirations from 22 to 24. On September 30th she was able to take nourishment and stimulants by the mouth, but the stools continued brownish and thin, and when an attempt was made to administer beef-tea or any food by the mouth, vomiting occurred. The cyanosis disappeared, and it was then observed that she was shrunken and emaciated. She continued to improve, and on the fourth day after admission was carried into the open air, where she received a sun-bath for two hours.

The following summary of the treatment may not be without interest. Upon admission she received a hot plunge bath. The first enteroclysis or intestinal injection was retained for three minutes, and afterward these injections were repeated every two hours. A subcutaneous injection of one pint of a $\frac{6}{10}$ per cent. solution of sodium chloride was given the day after admission. At first whiskey was administered by the mouth, but afterward ten minims were given hypodermatically every four hours. The total duration of this patient's illness was five days, and her convalescence was extremely rapid and uninterrupted.

The *third* case showed the typical symptoms of Asiatic cholera, and death occurred forty-one hours after admission to the hospital. The patient was a Russian boy, aged two years, who was admitted to the ward from the steam-

ship Bohemia at 2 P.M. He complained of pain, and vomited and purged freely. His face had the bluish and shrunken appearance that is so characteristic of this disease. The upper and lower extremities, as well as the face, were cold and cyanotic. The pulse was very weak, about 100 per minute, and the respirations were shallow, averaging 42 per minute. The temperature was 98° F. and soon descended to 97.4° F. He made no complaint of pain nor of muscular cramps. At first everything taken by the mouth was vomited, but soon this symptom subsided. The bowels moved frequently, the discharges were copious in quantity, whitish, and contained a large quantity of epithelium, giving them a rice-water-like appearance. The patient soon became apathetic, and during the night continued to purge and vomit. During the day the cyanosis became extreme, especially in the face and extremities. The remainder of the skin surface was whitish in color, and when a fold was pinched between the thumb and forefinger it remained elevated for some time, and then gradually resumed its former condition. The pulse became weaker, small, and increased from 90 to 100; the respirations rapid and shallow; and the temperature varied from 98.8° to 99.6° F. There was no vomiting, except that which was excited by attempts to administer food or remedies. During the night he had two copious choleraic discharges from the intestine. He continued in about the same condition until his death.

The spirillum of cholera was obtained from the early intestinal discharges. The autopsy was performed six hours after death. The cadaver presented the same general appearance as has been already described. *All* the tissues and *all* the organs of the body were remarkable for their extreme dryness. Both pleuræ showed extensive old adhesions, particularly on the left side. The lungs, liver, spleen, and peritoneum were normal, but *dry*. The kidneys showed acute parenchymatous nephritis. The entire subcutaneous tissue was dry and dark, and the skin when pinched remained elevated, the same as during life. A rectal tube was introduced for a distance of ten inches, and to this was attached an ordinary fountain syringe, the reservoir of which was placed at an elevation of five feet, and in this manner two pints of water were passed through the ileo-cæcal orifice, filling the small intestine and stomach.

Upon admission he was placed in a plunge bath having a temperature of 104° F., and calomel in gr. j doses was given every hour for three doses. One quart of a hot 1 per cent. solution of tannic acid was injected into the intestine every two hours, and also a subcutaneous injection of half a pint of a hot $\frac{6}{10}$ per cent. solution of sodium chloride, containing 1 per cent. of brandy. Every hour ♏v of whiskey were given hypodermatically, and at night oxygen was given by inhalation. All attempts to administer food or remedies excited vomiting.

The *fourth* case illustrates how rapidly death may occur in cholera. The patient was a Russian emigrant, aged eight years, admitted from the steam-

ship Bohemia at 6 P.M. on September 26th. She told us that in the morning, at 2 o'clock, vomiting and purging began, and between the hours of 2 A.M. and 6 P.M. she had three large rice-water discharges from the bowels and two similar discharges from the stomach. Upon admission she presented the characteristic facies of cholera. The orbits were deeply discolored, and there was a suffused reddish-purple blush over the arms, nose, legs, and feet. The cyanosis was especially marked on the left side of the neck, arm, and leg. This was due to the position assumed while on board the tugboat, which was used in conveying her from the steamship to the hospital. At first she was able to say a few words, but soon became unconscious. Her respirations were very shallow, numbering 35 to the minute. Her axillary temperature was 96.6° F. Soon after admission she vomited a moderate quantity of light-brownish fluid, and at 8.15 P.M. she again vomited twenty-eight ounces of a liquid having the same character. At 9.15 P.M. she discharged a large quantity of rice-water liquid per rectum, and she died at 10.15 P.M., or four hours and fifteen minutes after admission, and twenty hours from the appearance of the first symptoms of the disease.

Upon admission she received a hot plunge bath, and fifteen minutes thereafter her stomach was washed out with one pint of the solution of boric acid at the temperature of the body. At 8.45 P.M. she received an enteroclysis of two pints of the 1 per cent. solution of tannic acid at a temperature of 104° F., which was rejected. At 9 P.M. oxygen was used freely, and at 10 P.M. a subcutaneous injection of one quart of distilled water containing $\frac{6}{10}$ per cent. of sodium chloride and 1 per cent. of whiskey was administered. From time to time whiskey was given by the mouth. Oxygen was continued until her death.

The autopsy was performed eighteen hours after death. Rigor mortis was extreme, and the entire skin surface was dark-bluish in color, as were also the matrices of the nails. The body was considerably shrunken and emaciated. There was but little subcutaneous fat. Both pleuræ were free from adhesions. The lungs were collapsed, dark in color, but otherwise normal. The thymus gland was unusually large, measuring three by two inches. The right ventricles were contracted, and each contained a moderate-sized clot. The ventricles and auricles and cavæ were over-distended with blood, coal-black in color, and tar-like in consistency. The small intestine showed exfoliated epithelium and injection of the bloodvessels. The heart muscle and pericardial sac were normal. The liver was moderately enlarged, the right lobe being unusually flattened at its base, and its entire surface was mottled with irregular areas of a whitish, wax-like material passing into the substance of the liver. The left lobe of the liver was more uniformly infiltrated with the same material, but its consistency was normal. The gall-bladder was over-distended with a thick, dark-green bile, containing mucus. The kidneys were cyanotic, but otherwise normal. The spleen was normal in size, and its pulp was black in color and glutinous. The bladder contained about two drachms of pale urine. The gastric and intestinal discharges were

typical in appearance, and contained the spirillum of cholera. There was complete suppression of urine. The skin and subcutaneous tissues were so dry that when pinched the fold remained for several minutes.

The remaining cases were in many particulars like those already described; but before concluding this clinical description, I beg your indulgence while I narrate the history of the most interesting of all the cases observed, in which we were fortunately able to note minutely every change that took place from the beginning to the end. This patient exhibited nearly all the symptoms of a typical malignant case of Asiatic cholera; illustrating also, in an equally striking manner, the results obtained by treatment.

A male, aged twenty-four years, a native of Germany, was admitted to the Swinburne Island Hospital on September 27, 1892, at 11 A.M. His muscular and osseous systems were unusually well developed, and it was reported that he was perfectly well on the morning of September 27th until 4 A.M., when he first complained of pain in the abdomen, which was followed by two loose stools. He continued feeling well until we saw him at 10 A.M., when, in view of the diarrhœa, it was deemed wisest to remove him to the hospital, although his general condition did not indicate that he was suffering from cholera. He objected strongly to his removal, and said that he felt perfectly well. He walked from his berth to the side of the ship and down a rope ladder to the quarantine tugboat. He arrived at Swinburne Island and reiterated his statement that he felt perfectly well, and walked from the quarantine boat to the door of the hospital, when *suddenly* he complained of weakness in the knees, and fell to the ground in a state of collapse. He was carried to the ward in a condition of partial stupor, from which he was easily aroused. He responded to all questions in a manner showing that his consciousness was perfectly preserved. Soon he complained of agonizing cramp-like pains in the arms, feet, and knees, which recurred, more or less regularly, at intervals of thirty minutes. Immediately after his admission his countenance presented the typical appearance of cholera. The eyes were deeply sunken, the pupils were contracted to the size of a pin-point and surrounded by dark circles. The lips, cheeks, arms, hands, legs, and feet were cyanotic. The entire skin-surface was dry, and when the skin was pinched it remained elevated and compressed for several minutes. There was *no pulse* at the wrist. The temperature was 98.5° F., the respirations shallow and 18 per minute, and the voice whispering. I was unable to detect any special coldness in the expired breath. He was at once placed in a hot plunge bath having a temperature of 104° F., and was given four pints of a 1 per cent. solution of tannic acid at a temperature of 104° F.; also two pints of

distilled water at a temperature of 100° F., containing $\frac{6}{10}$ of 1 per cent. of sodium chloride and 1 per cent. of whiskey. He responded but slightly to this treatment; the pulse was scarcely perceptible, was filiform in character and about 100 per minute. At 11.30, or thirty minutes after his admission, he complained of intense agonizing pain in the legs, feet, and hands. These cramps forced the feet into extreme extension, and twisted and distorted the fingers. The pain was so great that it became necessary to administer a hypodermatic injection of one-eighth grain of morphine. The hypodermoclysis and enteroclysis were repeated. Camphor was also given hypodermatically. He inhaled oxygen constantly, hot bottles were applied to the extremities, and hot air was conducted under the bed-clothing from a large steam radiator. He now vomited a large quantity of a clear liquid and passed several copious rice-water discharges from the bowels. His collapse deepened; the pulse became slow, feeble, and almost imperceptible; and his respirations shallow. His intellection was clear, and his condition is best described by the word "terror." At 2.30 P.M., or three hours and thirty minutes after admission, he was extremely restless and anxious, and the choleraic intestinal discharges continued. He moved from side to side, tossing the arms about wildly and calling aloud for air. The hypodermoclysis was repeated and hydrochloric acid and brandy were administered by the mouth. At 5 P.M. he was pulseless and complained most bitterly of intense pain, produced by the tonic spasm of the muscles. These cramps were so violent that the muscles were knotted and felt board-like. At 6 P.M. the hypodermoclysis was repeated; the pulse was scarcely to be felt, and thirty minims of whiskey were given hypodermatically, and repeated for five doses, but with no effect. He now passed two copious liquid stools containing whitish shreds composed of intestinal epithelium, giving the discharges their rice-water-like appearance. At 6.30 P.M. he passed three more stools, and at this time we were of the unanimous belief that he must speedily die. At 7 P.M. he showed slight reaction. His extremities became a trifle warmer and the pulse more easily counted. At 10 P.M. hypodermoclysis and enteroclysis were repeated and a hypodermatic injection of twenty minims of whiskey was given every thirty minutes until 1 A.M. At 11 P.M. the intellect was clear; the eyes horribly sunken and surrounded by dark circles; the nose pinched; the face shrunken; the voice whispering and so feeble that he was compelled to *rest* between words in replying to questions. He now complained of pain in the chest and renal regions. His extremities grew warmer and perspiration was visible on the trunk. This symptom was peculiar and rare, as in none of our cases was perspiration visible. The mental condition—terror—persisted. At this time he ejected large quantities of rice-water liquid by five acts of emesis. At 1 A.M., September 28th, this slight improvement continued, and the hypodermoclysis and enteroclysis and hypodermatic injections of whiskey were repeated. At this time he had three movements of the bowels, the discharges being composed of the tannic acid solution given by in-

jection. At 5 A.M. a hypodermoclysis and a subcutaneous injection of whiskey were administered, and at this time the improvement was quite marked and he slept quietly for a few hours. At 8.30 A.M. the enteroclysis was repeated and the whiskey suspended. At 9 A.M. his physiognomy underwent a truly remarkable change for the better, becoming slightly flushed; the expression about the eyes became more natural and the ghastly deathlike pallor had disappeared. The entire skin-surface, especially that of the extremities, was warm, and the pulse was of full volume, soft in quality, regular, beating 100 per minute. At 11 A.M. it seemed incredible that such wonderful changes could have occurred in the twenty-four hours which had just elapsed. At 10.30 A.M. the enteroclysis was repeated, and at 10.50 A.M. the pulse was full, regular, soft, and 88 per minute; the respirations were normal, and the expression good. He responded intelligently to questions, and the improvement continued.

In both flanks where the needle had been inserted repeatedly for the subcutaneous injection of the sodium chloride solution, the tissues were hyperæmic and sensitive to the slightest touch. No extravasation of blood occurred, and in three days these symptoms disappeared. At no time was a single drop of urine excreted. In ordinary cases the rate of absorption, after the hypodermoclysis, varies between forty and sixty minutes, whereas, in this case three hours were required, thus showing that the power of absorption had been almost abolished. So soon as the liquid was absorbed a second enteroclysis was given. Hypodermatic injections of twenty minims of whiskey were repeated almost hourly until midnight. He passed a good night, vomiting but twice, and had dark-colored stools. His general condition remained unchanged. On September 29th he received champagne and now recognized that he was convalescent. He was given whiskey and Seltzer water, equal parts, every three hours, and at 10.45 A.M. for the first time he passed one pint of urine, precisely forty-eight hours after admission. The examination of this urine showed a distinct trace of albumin; no sugar; sp. gr. 1024; acid reaction; and the sediment showed numerous granular tube-casts. The liquid movements continued every two hours; and again he passed a normal quantity of urine. On September 30th the diarrhœa continued to average one stool every two hours, the quantity passed was small and the color had become dark. The patient was excessively irritable and nervous. He improved slowly for two days and then sank into a typhoid state.

There was no enlargement of the spleen or elevation of temperature.

As we feared, but little improvement occurred and the typhoid state continued until death, which took place on October 5th, or eight days after his admission to the hospital.

The post-mortem examination revealed the ordinary changes that are found in cholera, and the kidneys showed a severe grade of acute parenchymatous nephritis.

Mortality.—Of the 11 cases of cholera under my personal observation, 8 recovered and 3 died, which gave a mortality of 27 per cent. As one of these cases died two hours after admission, it could be fairly excluded in giving the percentage mortality of cases under treatment, and would reduce this mortality to 18 per cent.

The total number of cases admitted to the Swinburne Island Hospital was 72, and the number of deaths was 20, or a general mortality of 27 per cent. In addition, 56 suspects were admitted, of whom 46 presented the prodromic symptoms of cholera. If these were included our mortality would be reduced to 17 per cent. The mortality from all cases *under treatment*, including the 46 suspects, and excluding the deaths occurring within two hours after admission, and those deaths occurring later from complications, would be 11 per cent.

The mortality in cases of cholera that occurred on shipboard varied between 50 and 98 per cent., while the percentage of deaths in Hamburg varied between 50 and 60 per cent.

In considering the number of deaths with relation to the question as to how far the treatment was responsible for this low rate of mortality, it is well to remember that most of our patients were half-starved, rhachitic, and anæmic Russian emigrants, many of whom were children; that they were in a specially weak condition, due to the long voyage and also to the fast days which their religion imposed upon them; that the premonitory stage in the epidemic was present to a slight degree, or not at all; that many died before the treatment could be inaugurated, and that this epidemic was especially virulent.

Despite these unfavorable circumstances our general mortality was but 27 per cent., while that of Hamburg was from 50 to 60 per cent. After a very careful deliberation, in which we discussed everything that could have influenced the mortality rate, we were convinced that the difference was chiefly due to the method of treatment employed.

Treatment of Cholera.—As to *prophylactic treatment*, the rules found necessary were both few and simple. Each of us wore a separate suit of clothes for the hospital, and we avoided *physical contact* with patients except when it became necessary. Regularly, *each time after examining a cholera case*, we would wash the hands thoroughly with hot water and soap, afterward immersing them in a 1 : 500 solution of bichloride of mercury. The convalescents and those employed about the hospital were instructed to drink a "lemonade" of ten drops of hydrochloric acid to the glass of water. No ordinary water was used, and we drank exclusively of carbonated distilled water or Apollinaris water. All food was thoroughly cooked, and green vegetables, salads, and indigestible foods were avoided. These precautions were so effective that, despite the dense ignorance of the people under our care, during the entire epidemic but two cases of cholera originated on Swinburne Island. In both instances these emigrants drank red wine until they became thoroughly intoxicated, and this was followed by acute catarrhal gastritis and infection by the spirillum of cholera.

In the treatment of the *stage of collapse* the patient was first immersed in hot water, and then given a subcutaneous injection of a quart of hot, sterilized water, containing $\frac{6}{10}$ per cent. of sodium chloride and 1 per cent. of brandy. This procedure is known by the name of *hypodermoclysis*. The first injection in an adult may be one or two quarts. In but one of our cases did an abscess form. Usually the only complaint made when this operation was frequently performed was sensitiveness and pain on light pressure over the region of the punctures. In favorable cases absorption takes place in from thirty to forty-five minutes, but in cases of collapse approaching death, so long as four hours may be required. It therefore becomes evident that the rate of absorption is of great prognostic importance. The best position for these subcutaneous injections is in the flanks, in the region of the floating ribs, in the median axillary line, although they may be administered in the buttocks or inner aspect of the thighs. The

neck is to be avoided, as there is danger of producing œdema of the glottis. Ordinarily, a hypodermoclysis may be repeated every two hours, and in severe cases it may be well to inject one quart in *each* flank, repeating the injection so soon as it has been absorbed. In supplying the indication for heat it is necessary that the solution should have a temperature of about 104° F., and care should be taken that it is thoroughly sterilized immediately before it is introduced beneath the skin. In each of our cases an ordinary Davidson syringe was employed, but I would strongly urge the adoption of the ordinary fountain syringe, as by this means the liquid may be slowly introduced, and hydrostatic pressure controlled to a nicety by raising or lowering the reservoir. It requires from twenty to thirty minutes to introduce one quart of liquid, and usually it is unnecessary to attempt to disperse the liquid by manipulation, though it may be advisable when, in grave cases, rapid absorption becomes necessary. As one would naturally suppose, this subcutaneous liquid forms a large oval swelling, the size depending upon the amount of liquid that is introduced.

A good working rule regarding the quantity of the injection is the following: For an adult, two pints; for adolescent, one pint; for an infant, one-half pint.

An *enteroclysis* consists of a 1 to 2 per cent. solution of tannic acid, having a temperature of 45° C. or 113° F. For an adult, two quarts may be given; for an adolescent, one quart. This solution should be introduced very slowly, and in the treatment of each of our cases the Davidson syringe was employed, but I should advise the use of a medium-sized soft-rubber rectal tube, having one outlet one-half inch from the extremity, and a second on the opposite side, two inches from the extremity, and the terminal portion closed so as to facilitate its introduction. In an adult this tube should be well oiled and gently and slowly introduced by a slight rotary and inward pressure for the distance of ten inches. To this tube should be attached an ordinary fountain syringe, the same as the one suggested for hypodermoclysis. The advan-

tage of this method is that the hydrostatic pressure may be modified immediately to suit the particular case in question, and in this manner the rate of discharge of the liquid may be regulated. It is necessary that the solution should be *slowly* introduced, occupying not less than *ten minutes*. The tube should then be slowly withdrawn, and gentle pressure applied to the anus in an inward direction toward the anterior perineum. The patient should be encouraged to retain the liquid as long as possible. Not infrequently if the first desire to void the injection is overcome, it is retained without further difficulty.

It has been hitherto generally believed that the ileo-cæcal valve prevented the entrance of liquids from the colon into the ileum. In many of our cases, after giving an enteroclysis of tannic acid we were convinced that the liquid entered the small intestines, and this opinion was strengthened when several of our patients *vomited* this same solution.

In order to investigate this matter more carefully, the following experiments were made:

A fountain syringe containing three pints of water was suspended at an elevation of five feet, and a rectal tube introduced for a distance of six to ten inches.

Case I.—Male child, aged two years; dead of cholera; the liquid passed readily, filling the intestines and stomach.

Case II.—Male child, aged two years; dead of marasmus; liquid passed freely, filling the intestine and stomach.

Case III.—Child, aged six years; dead of measles; the liquid passed readily through the entire intestinal tract, and flowed from the mouth and nose.

Case IV.—Child, aged three years; dead of measles; the liquid passed readily through the ileo-cæcal orifice, filling the small intestine.

Case V.—Child, aged three years; dead of measles; the liquid refused to pass. A post-mortem examination showed that the colon was over-distended and that there was a twist in the ileum against which the distended colon pressed, rendering it impossible for a liquid to pass into the ileum.

Case VI.—Female child, aged eighteen months; dead of measles; the liquid failed to pass both before and after opening the abdominal cavity. In this case the ileo-cæcal valve was small and the lips of the valve were in

close apposition, rendering it impossible for any liquid to pass from the colon into the small intestine.

Case VII.—Child, aged two years; the liquid refused to pass, and, upon examination, the ileo-cæcal valve was competent.

These observations show that in two cases the valve was competent to prevent irrigation of the small intestine, and in one case, owing to the peculiar twist in the ileum and the pressure of the over-distended colon, liquids failed to enter the ileum. This case is particularly instructive, and shows that in a certain number of cases success may be looked for, even though the first attempt prove unsuccessful. In four cases there was no difficulty whatever in the passage of liquids from the anus to the stomach, or even out through the mouth and nose.

As *coldness* of the body and *lowering* of the *central temperature* is an almost constant condition in Asiatic cholera, it becomes necessary to supply heat. This is accomplished by heating the liquids used in hypodermoclysis and enteroclysis, and also by the hot plunge bath, which is always given to the patients in the state of collapse. I should further advise that the entire skin-surface be covered by soft woollen under-garments, and from time to time hot air should be conducted beneath the bed-clothing. Advantage may also be taken of hot-air bags and hot bricks. The patient should be covered by two woollen blankets and a counterpane. A most excellent practical suggestion to add to the heat of the body has been made by Dr. Francis X. Dercum, namely, that the patient be placed upon a water-bed, through which hot water should constantly circulate.

Stimulation. The best method of administering stimulants is by deep hypodermatic injections of whiskey, repeated every two hours or less frequently, according to indications. For an adult, twenty minims may be employed; for an adolescent, ten minims; and for an infant, five minims. During the stage of collapse, if there is any tendency to vomiting, it is wisest to avoid the administration of *any* substance by the mouth.

Lavage. As the stomach frequently contains large quantities of choleraic liquid, it is often advisable that a soft-rubber stomach-tube be introduced, and that lavage be thoroughly performed, using hot tannic acid solution as in washing out the intestines.

Hydrochloric Acid. As the growth of the spirillum of cholera is inhibited in acid media, and as hydrochloric acid is the normal acid of the gastric juice, it is desirable that it be administered, diluted in water, at intervals of two hours. An adult may receive fifteen minims; an adolescent, ten minims; and an infant, five minims, taken slowly in sips in a glass of water.

NOURISHMENT.—*Liquids.* The only nourishment that should be administered is peptonized milk, or milk that has been sterilized, in small quantities, about two ounces every two hours. If this is not well received, it may be surcharged with carbonic acid gas, or koumiss may be substituted. Iced champagne, in small quantities, may also be given, or iced wine-whey made in the proportion of one part of sherry wine to four parts of milk. The only liquid that should be permitted is carbonated distilled water. When vomiting is persistent, all attempts to administer remedies or food by the mouth should be avoided.

In conclusion, I cannot refrain from tendering my hearty thanks to Drs. Byron and Abbott, of the Loomis Laboratory, New York, to whom I am indebted not only for the opportunity of making this clinical study, but also for the many courtesies which I have received at their hands; and further, to Dr. Byron is due the credit of having initiated the treatment of these cases in the manner herein pointed out.

To Dr. Jenkins, the Health Officer of the port of New York, my thanks are due for his many kindly courtesies, and for the aid which at all times he gladly extended to me.

DISCUSSION.

Dr. Francis X. Dercum: In regard to the conservation of heat, a device which has been used by myself in the treatment of collapse for a number of years past, and also by Dr. Wood, would here doubtless prove of great service. It is to fill an ordinary water-bed with hot water.

Tannic acid could also be administered by the mouth, in addition to its administration by the bowel. I would suggest that it be given, dissolved in petrolatum, in large, soft gelatin capsules. These could be swallowed without discomfort, and would doubtless have an antiseptic action as regards the cholera bacillus.

Being dissolved in petrolatum, the absorption of the tannic acid would be largely prevented and its action restricted to the *mucous membranes* of the stomach and intestines. Finally, it would be possible to make the solution much stronger than one per cent.

Dr. George E. Shoemaker: One point in the technique of intra-venous injections that I have observed in cases of intense acute anæmia, as from hemorrhage, is that the position of the vein may be sometimes detected by a little depression in the position where there should be a fulness. I have also noticed in the use of the long gravity-tube, a decided cooling of the quantity of water contained in the tube. It is, therefore, important to immerse the whole tube in a large vessel of water at the desired temperature until you are entirely ready to start the current. Otherwise the first flow of water may be at a temperature of 70° or 75° instead of 104°.

Dr. Charles Hermon Thomas: In 1860, I had an extensive cholera practice. I found that hot salt-bags were the most available things that I could depend upon in the houses where cholera was apt to occur. A quart or so of salt was put in a pan on the stove, and, when heated, put in bags, stockings, or anything else that was to be had, and packed around the patient. I also used, with great satisfaction, hot oat-bags and hot meal-bags.

Dr. H. Y. Evans: In regard to prognosis, it has been asserted that as soon as you see a dark stool, you may expect the patient to get well. I should like to know if this was observed in the cases reported.

Dr. Frederick P. Henry: My remarks will be only of an indirect application to Dr. Daland's intetesting paper, because I have treated no case of cholera either by enteroclysis or hypodermoclysis. I wish to call especial attention to the fact that cholera is not the only disease in which the tissues are in urgent need of water, although it is undoubtedly the one in which that need is most imperative. Typhoid fever is one of the diseases I have in mind, and I am sure that many Fellows of the College recollect the emphasis and eloquence with which the late Dr. J. F. Meigs insisted upon the necessity of supplying typhoid patients with an abundance of water. I believe that the tissue degenerations of typhoid fever, which are usually attributed to excess

of heat, are quite as likely due to deficiency of water. The desiccation and decay of vegetable tissues during the torrid heat of summer are universally attributed rather to deficient water than excessive heat, and it seems to me surprising that analogy has not caused the same opinion to prevail with reference to animal tissues.

The readiest and most effective method of supplying the tissues with water is certainly hypodermoclysis. Several years ago I treated a number of cases of pneumonia at the Philadelphia Hospital by injecting under the skin a solution of common salt of the same strength as that employed by Dr. Daland in his cholera cases; in round numbers, 50 grains to the pint. I was induced to employ this method because, in the cases in question, the system of the patients seemed to be in urgent need of water; this need being chiefly indicated by a dry, parched tongue, and constant thirst. The results were most gratifying, several grave cases showing immediate improvement and ultimately recovering.

Since the time to which I refer, Dr. Hermann Sahli[1] has treated several cases of typhoid fever by hypodermoclysis and with most encouraging results. As I have elsewhere remarked: "One of the most rational indications of typhoid fever is certainly fulfilled by such injections."

I wish to indorse Dr. Daland's statement as to the ease with which a saline solution may be introduced beneath the skin and the rapidity with which it is absorbed; although the method is so widely practised abroad that such indorsement is scarcely necessary.

Dr. Daland: The method of supplying heat mentioned by Dr. Dercum would undoubtedly be of great advantage whenever it could be employed.

With regard to the prognostic value of dark stools, I would say that we noted that where cases improved, the color of the stools became darker. This symptom is therefore of good prognostic omen, but the one to which we attached the greatest importance was the appearance of the first drops of urine. So long as no urine was excreted the case was looked upon as one likely to die. Again, when the liquid injected beneath the skin is quickly absorbed the prognosis is far better than when it remains for three or four hours.

[1] Volkmann's Sammlung klinischer Vorträge, No. 11, 1890.

A COMMON PIN IN THE VERMIFORM APPENDIX, WITH SECONDARY ABSCESS OF THE LIVER.

By GEORGE ERETY SHOEMAKER, M.D.

[Read November 2, 1892.]

This specimen was removed *post-mortem* from the body of a man who had had abscess of the liver and general peritonitis. It is exhibited for two reasons: First, because it is uncommon to find a genuine foreign body in the appendix, nearly all bodies so reported being fecal concretions. Second, to suggest the interesting point of probable relation of abscess of the liver to the pin by retro-peritoneal infection of a region drained by the portal system. The coincidence of hepatic abscess with appendicitis is quite rare.

The man died about thirty hours after operation for pericæcal abscess and obstruction of the bowel, with fecal vomiting. The presence of the hepatic abscess was unknown, nor was there anything to indicate its existence, while the symptoms from obstruction were urgent. From an operative point of view the case is not of great interest, as it was one of those which are first seen so late in the disease that operation can hardly save. Indeed, could it have been possible to know the conditions present, no operation would have been done. I did not see the patient until within an hour of the operation, and I am, therefore, dependent upon the kindness of Dr. Buck, resident physician at the Methodist Hospital, for the notes of his previous history, which was very meagre.

He was eighteen years of age, a native of Philadelphia, and had been ill about twelve days, chiefly under home treatment. He was admitted to the medical ward under the care of Dr. Hollopeter, who transferred him to the surgical department. The trouble began with a chill and coincident pain in the right hypochondrium, which, his sister since states, he then referred to as "that old pain." While in the hospital, however, he did not admit any previous disorder. When I first saw him he had had complete obstruction of the bowels for five days, and obstinate constipation for several days before that. He had had fecal vomiting and subnormal temperature for twelve or fifteen hours, with evident peritonitis. The abdomen was tense, and was œdematous, dusky, and more resistant in the right flank

though, from the great tension of the walls, the presence or absence of a tumor could not be made out. The breathing was thoracic, the temperature 95.6° F., the pulse 90 or below. The mind was clear, though there had at times been delirium. There was no pain, but a gulping up, rather than a vomiting, of a thin brown offensive fluid with decided fecal odor. Well-applied efforts to move the bowels had failed. There was no evidence of liver disease, no jaundice, soreness, redness, venous obstruction, or enlargement of the organ, while there was evident obstruction of the bowels, with peritonitis, which the dusky and œdematous appearance referred to the right flank. Incision in the right semilunar line disclosed pus and thick yellow lymph as soon as the peritoneum was cut. Two or three ounces of pus were removed from a cavity limited outside by the pelvic wall and inward by adherent coils of intestine. It was inside the peritoneum. The intestines were tightly distended, and as adhesions appeared to limit the pus cavity on all sides, it was not considered wise to search for and remove the appendix, which could not be readily felt. Taking into consideration the man's condition, such an attempt at this time would have been highly dangerous. The abscess cavity was flushed with boiled water and packed with iodoform gauze about a glass drain, the incision being left open. This procedure gave great relief. The temperature rose to normal, and in a few hours reached 99.8° F. The vomiting and hiccough stopped at once, and large quantities of urine began to be secreted, no less than eighty-seven ounces being obtained by catheter, at short intervals, in the next twenty-four hours. The improvement did not continue, however, beyond this period. The bowels did not move, and the patient sank very rapidly in the evening of the day following the operation, with a very weak pulse, never over 120, and a temperature never above 100.6° F.

The necropsy showed the cavity at the seat of operation sweet and clean. The parietal surface of the great omentum was bathed in pus, which had apparently come from a ruptured abscess of the liver. There was no pus upon the intestines, but a recent general peritonitis had caused slight general intestinal adhesions, and the widespread deposit of a fresh translucent coagulated lymph, which immediately below the liver and in the left flank was yellowish and in solid masses.

The cause of obstruction had been the massing of convolutions and contained scybalæ in the left pelvis and pressure from above by distended intestine. Free purging had occurred soon after death, so that it is quite likely that the bowels would have moved had the patient lived longer. This, however, would not have saved his life, as he must have died from general purulent peritonitis from rupture of the hepatic abscess. The appendix extended downward toward the true pelvis, and though surrounded by recent plastic deposit, was apparently not perforated, nor was it gangrenous. It contained a common pin, head downward. There was pus behind the peritoneum, along the front of the spinal column from the root of

the mesentery upward, extending a little way between the layers of the gastro-hepatic omentum and surrounding the ducts and vessels at the transverse fissure of the liver. This organ contained within its substance an abscess apparently acute, situated near the surface of the right lobe at its highest point. The pus, which was yellow, occupied a single irregular cavity about 2 by 2 by 3 inches in diameter. The liver was easily separated from the diaphragm, pus appearing as soon as this was attempted. As has been said, the front of the great omentum was covered with fluid pus, which had not yet reached the intestinal surfaces in visible quantities. It is probable that the general peritonitis present was caused by the rupture of the hepatic abscess and leakage in front of the liver after the operation; for had the whole attack, including the obstruction of the bowels at the time of the operation, been due to general peritonitis from rupture of the hepatic abscess and not to a localized peritonitis, it seems improbable that merely washing out the circumscribed area, which was shut off in the right side, would have caused the marked improvement that was noted for the first twenty-four hours.

The patient had been ill nearly two weeks, and may have had previous trouble in his right side. Was not the pin the original cause of the hepatic abscess? Could it not have pierced with its point through appendix wall and parietal peritoneum, and have infected the retro-peritoneal tissues, the veins of which belong to the portal system, although the lymphatics lead to the left subclavian vein by way of the thoracic duct? The line of retro-peritoneal inflammation would make this probable. The intra-peritoneal abscess operated upon is easily accounted for by the presence of the pin. The abscess of the liver was secondary, but not strictly pyæmic, according to my interpretation, since it was single and pus foci were not found in the kidney, spleen, or lung. The heart was normal.

REMARKS ON CERTAIN INDIAN SKULLS FROM BURIAL MOUNDS IN MISSOURI, ILLINOIS, AND WISCONSIN.[1]

By DANIEL G. BRINTON, M.D.,
PROFESSOR OF AMERICAN ARCHÆOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

[Made November 2, 1892.]

This series of interesting specimens is of historical as well as of anthropological interest. The first deformed American skull that was ever described came from exactly the region from which these came. It was described by Blumenbach in 1789. It was exhumed opposite St. Louis, on the Illinois shore.

Most of the skulls that you see before you are deformed skulls. The study of deformities in the American skull is truly a rich field. Of all the races of men, no one race has so given itself up to the artificial deformation of the skull as has the American. The subject of cranial study has been enriched by a work, that has reached Philadelphia within the past week, by Virchow, entitled *Crania Ethnica Americana*. It may be classed with the well-known work by Morton as the second complete exhaustive treatise, bringing the science up to date in this department of craniology. I am proud to say that Virchow repeatedly refers to the work of Morton as a work most meritorious in its day, and handing down to all craniologists many true conditions of

[1] Dr. Hinsdale announced to the College a donation to the Mütter Museum of sixty Indian skulls, presented by Dr. S. Weir Mitchell, to whom they had been given by Dr. George J. Engelmann, of St. Louis, Missouri. They were exhumed by Dr. Engelmann and Dr. Wislizenus from cysts near French Village, Illinois, eight miles southeast of St. Louis; from New Madrid County, Missouri, and from Geneva Lake, in southeastern Wisconsin. Seven of the skulls were from the big mound in St. Louis, now completely levelled.

The bones were found in single graves at a depth of two or three feet, and they had been buried probably after decay of the flesh. Some of the cysts in which they had been placed were lined with flat pieces of limestone. Dr. Brinton's remarks were made at the request of the President.

accurate science. Of course, as we might expect, a half-century has obliterated many of the ideas which Morton advanced.

The characteristics of these specimens is in their deformations. Most of the skulls present decided abnormalities, artificial in character. The deformities of the American skull have been classified by a number of authors, not by any means agreeing with one another, and, in fact, I may say that scarcely two agree. We may, however, assume certain general principles. There are two deformities, not intentional, though marked. We do not consider flattening of the occiput as intentional. Those who have been among Indians will have observed the method by which the papoose is carried on the back of the mother, usually strapped to a board, and in the flexible condition of the bones of the skull this brings about a flattening of the occiput. Morton was not entirely clear that this was an artificial deformity. In his lectures, and in some instances in his work, he classes flattening of the occiput as a characteristic of the American race. We, however, do not consider that it is, strictly speaking, an artificial deformity. It is accidental, not intentional. Nor do we consider the flattening of the front, in most cases, a deliberate deformity. It is also unintentional. The papoose, in addition to having the back of the head against the board, is secured to the board by a band passing around the front of the head, and this leads to flattening of the frontal region. These deformities are not considered in any sense intentional, but there are at least six others which have been carefully catalogued, and which are artificial and deliberate. This brings about these curious shapes of skulls, one of which Morton called the cuneiform, another the mitre skull, resulting in those excessive brachycephalic skulls which we see in the natives of the Lucayan Islands, where we find some skulls with a biparietal diameter of 90 to 100.[1] These are undoubtedly brought about by antero-posterior compression, with the intention of making the head very broad and short. The very short heads are found principally in the Lucayan Islands. There are two extreme forms, the short skull and the extremely long skull, where pressure is made laterally. Glancing over this collection, I do not find a fair type of the latter. They are found in two parts of the continent very remote from each other, to wit: in Peru and among the Flatheads of the northwest coast. These two nations, evidently without any communication with each other, developed the same ideas of the beautiful as regards the shape of the head. The characters of the deformities of the skull are closely localized throughout America, and when we see a deformed skull we can, as a rule, refer it to certain areas. I should have been somewhat surprised to have found a typical case of a long skull in this collection, for we do not find them in the locality from which the collection comes. Bartram, who lived, about 1790, among the Chickasaws, states that the braves have high foreheads and a small back of the head, and he regarded this as a characteristic of the race. It is, however, artificial. We do not find the low

[1] Percentage of occipito-frontal diameter.

and retreating forehead that we find among the Incas of Peru and the Chinooks of the northwest coast.

The skulls before us are, as a rule, typical of what we call southwest skulls. This division includes the skulls of the Choctaws, Chickasaws, Natchez, and, to some extent, the tribes north of them. The nations who inhabited the locality from which these skulls came must have derived the practice from the tribes south of them, which they could do by interchange of women.

Several questions come up in considering this subject. One is, Does this flattening, which is extreme in some cases, make any difference in the cubic capacity of the skull? An investigation of the cubical capacity of these deformed skulls shows that there has been a compensation, and that the capacity of the skull is not materially or not at all interfered with. Many range above 1500 cubic centimetres.

Again, does this displacement of the contents bring about any alteration in the intellectual capacity? The answer to this is likewise in the negative. As I have before observed, we find in Peru probably the most extraordinary cases of cranial deformity anywhere in the world, and yet it is a familiar fact that the Incas stood at the high-water mark of intellectual capacity.

Another question is whether these deformities, which have been continued generation after generation for many centuries, have exerted any influence in the way of transmission of the peculiar form of skull? This subject is very interesting in reference to some recent biological studies. The opinion has finally drifted toward the conclusion that there is no tendency whatever to the transmission of these deformed skulls, no matter for how many generations the practice has been continued.

These deformities did not belong to the American race originally. The old skulls are not deformed either in North or South America. When we go far back to the time when many of the now extinct mammalia were still extant, we find the skulls normal in character. Evidently, therefore, the method of carrying the children which brought about these unintentional deformities was learned after the American race had been on this continent for a long time.

In conclusion, I would say that the general character of the American cranium, wherever you see it, has been said by Virchow to be uniformly high. We do not find among them skulls presenting any anthropoid or theramorphic characters, as in the oldest skulls of Europe. There may be some exceptions which have not as yet been brought before the eye of science. There are alleged to be such. We, however, do not find the characters seen in the old European skulls. The conclusion that morphologists have arrived at is that the human race appeared first in America at a time when man was developed much higher in Europe than at the oldest time that we can trace him there.

FRACTURE OF THE INTERNAL CONDYLE OF THE HUMERUS TREATED BY EXTENSION; SUIT OF MALPRACTICE; VERDICT FOR DEFENDANT.

By C. E. KURTZ, M.D.,
BELLAIRE, OHIO.

[Presented by Dr Oscar H. Allis, December, 1892.]

History of the Case.—On the 7th of May, 1889, I was called to see a boy about nine years of age. He had been thrown down by his playmates and fallen upon during school recess. I saw him about fifteen minutes after the accident, and, upon examination, found an almost vertical fracture through the trochlea of the left humerus. In flexing the forearm there was great lateral displacement of the internal condyle, which almost disappeared during extension. I therefore dressed the arm in the extended position, a method of treatment introduced to the medical profession by Dr. Oscar H. Allis,[1] of Philadelphia, and one that I have repeatedly employed with most excellent results for many years. The dressing consisted of a layer of cotton extending nearly the length of the arm and forearm, and most abundant about the elbow. This was covered in by means of a muslin bandage. Upon the latter was smeared an albumen paste (made from the white of eggs and flour), after which a second layer of muslin bandage was applied.

I saw the boy seven times at his home from the day of the accident (May 7th) to May 15th. On the 15th I re-dressed the arm in extension, at which time there was swelling and great discoloration from extravasated blood. This showed that there was not only a fracture of the condyle, but also great injury to the soft tissue, arteries, nerves, etc. From the 15th of May to the 4th of June, I visited the boy seven times at his home. On the 4th of June, or twenty-eight days after the fracture, I removed the dressing and made the first manipulation with the arm—that is, I gently bent the arm until pain ensued. The pain was so great that the boy fainted. There was then only slight motion of the elbow-joint; prona-

[1] Annals of the Anatomical and Surgical Society, Brooklyn, vol. ii., No. 8, 1880.

tion and supination were good. I then made passive motion with the arm at his home on the 10th, 14th, and 18th of June, 1889, but only bending the arm to such a degree that it caused him no pain. After the 18th of June, I requested the patient to come to my office for passive motion. He came two or three times. About six weeks after the fracture I removed the dressing, and re-dressed the arm with roller bandage only. I directed the mother to make passive motion daily, instructing her how to do it, and to occasionally bring the patient to my office, so that I myself could make passive motion. The mother came about the seventh week without the boy, saying she was not satisfied with the progress of the arm, and thought the arm should have motions at that time. *She asked me to take the boy to some "good doctor" to have the arm "fixed right."* To this I replied that she was entirely in too great a hurry about it, and that the boy had then a better arm than I had promised on the day of the accident. I will here state that it is my invariable rule to make to my patients at the time of the accident a full statement of the danger of this class of injuries. In this instance I pointed out the probability of ankylosis in a fracture extending into the elbow-joint. As the parents failed to bring the child to my office, I made it my business to see him at his home. I found him but two or three times, and strenuous objections were raised to my making motions with the arm, owing to the pain which it caused. On the 18th of July, a few hours after I had made a morning call, only to find the boy again away from home, I received notice from an attorney to adjust the damage, otherwise I should be sued. To this I made no reply.

At the beginning of court in September, I received notice from the clerk of court that suit had been entered against me, the plaintiff claiming five thousand dollars damages for neglect, unskilful treatment, and causing great pain.

The case came to trial March 14, 1890, Judge J. D. Briggs upon the bench.

Before the trial I made (at the suggestion of Dr. S. S. Thorne, of Toledo) application to have the boy's arm examined by all the medical witnesses for defendant, as well as for plaintiff. This was granted me. Sixteen physicians were present, six for plaintiff and ten for defendant, and their surprise was great to find an arm with hardly any deformity. When the boy stretched out both arms one could not determine which was the broken arm, there being no "gun-stock" deformity such as we have, as a rule, when treated in the flexed position. The only way one could tell which was the injured arm was by the

partial immobility of the elbow-joint of the left arm. The arm, which had one-fourth of a flexion at the time I last saw him—July, 1889—was now almost stiff, *the lad being forbidden to move the arm at all, in order to make a good case.* The examinations of those physicians who were present showed: 1. That there had been a fracture of the trochlea near and close to the articulation of the head of the radius. 2. Coaptation had been good. 3. That there was now existing fibrous adhesion, and five degrees of motion. 4. Fairly good motion could yet be obtained by proper treatment.

The trial began with three medical witnesses for plaintiff, all of whom testified in favor of defendant; at the conclusion of which the plaintiff rested his case. The defendant called six medical witnesses.

The boy extended his arm to the jury while on the stand, but no one at that distance could tell the injured arm from its fellow.

The judge then charged the jury[1] as follows:

"*Gentlemen of the jury:* On the 7th day of last May, the plaintiff—Howard Whitney—a little boy, ten years of age, broke and fractured his left arm, and greatly injured it in other ways. On that same day Dr. Kurtz, the defendant—who then and there held himself out to be a physician and surgeon—was called and employed to set and reduce the fracture, and to attend and care for the injured boy until his wound was healed.

"Now, gentlemen, the plaintiff, by his next friend, claims that the defendant, the doctor, accepted this employment and undertook to set said bones and reduce said fracture, and to give to the injury such care and attention as were necessary to effect a cure; but the plaintiff claims that the defendant so negligently and carelessly reduced such fracture and set such bones and cared for such wound, that the boy's arm has become deformed and stiffened—so that it is now useless.

"The defendant admits that he is a physician and surgeon,

[1] The charge is quoted in full, simply because the case is the first malpractice suit arising out of the special method of treatment adopted.

and was such at the time of the injury; he admits that he holds himself out to the public as a physician and surgeon, but he denies that he was guilty of any negligence or carelessness in attempting to cure the boy's arm.

"Every person who offers his services to the public generally, as a physician and surgeon, impliedly contracts with those who employ him that he is a person of the skill and experience possessed ordinarily by those who exercise that profession.

"When the defendant here, Dr. Kurtz, accepted this employment, there was an implied contract between the father of the boy and the defendant, Kurtz, that he—Kurtz—was then in possession of the ordinary skill and experience of physicians. The defendant, Kurtz, was not required, in attempting to reduce this fracture and set these bones and to care for this boy, to exercise the *highest* degree of skill and care and diligence, but the law only required of him the exercise of an *ordinary* degree of care and skill—that ordinary degree of care and skill that is usual in that profession; and, further, he is only required to exercise the ordinary skill and care of his profession, in that vicinity—not the usual ordinary care, skill, and experience that is usual at some other point, but the ordinary skill and care of a physician usual in the city of Bellaire, where the defendant was practising his profession.

"When Dr. Kurtz accepted this employment, he did not insure a cure; he did not assume, by the terms of his contract, that he would *cure* the arm, or that the arm should not be deformed or stiffened, as it now appears to be; but he only contracted with plaintiff that he *would attempt to make a cure*, and that in that attempt he would exercise the ordinary care and skill of his profession.

"If, gentlemen of the jury, in attempting to set these bones and to reduce this fracture and to give the arm such attention as was required, Dr. Kurtz exercised the ordinary care, skill, experience, and diligence that was usual in his profession, the plaintiff cannot recover—notwithstanding you may find that the arm is deformed, or that it is now stiffened and useless.

"Now, Dr. Kurtz undertook to attend to the boy, and if, in

attending him and doing what was necessary in the premises, he did not exercise such care, such skill and such diligence, and such attention as was usual in his profession, then gentlemen, in that case, the plaintiff would be entitled to recover.

"The defendant, Dr. Kurtz, would not be liable for mistakes. If you should find from the evidence that he made mistakes of judgment, that fact, alone, would not warrant you in finding a verdict for the plaintiff.

"If the defendant exercised ordinary care—the ordinary care of his profession—and if, in the practice of his profession, he made a mistake in judgment, and you further find, at the same time, that he exercised the ordinary skill and judgment of his profession, and find that the defendant while in the exercise of such ordinary skill or judgment, was mistaken; or that his work would have been done in a different manner had it not been for his mistake in judgment, and a cure might thereby have been effected, yet, in that case, the plaintiff would not be entitled to recover for such mistake.

"It is claimed by the plaintiff that the arm ought to have been set in a flexed position; that it was dressed in the extended form. It is further claimed by the plaintiff that if it had been dressed in the flexed position, that, even if the arm had become stiffened afterward, it would have been of more service to the plaintiff.

"Gentlemen of the jury, in considering this question, you are to determine whether the doctor acted with the ordinary judgment, skill, and care requisite in his profession.

"Look to all the testimony, gentlemen, in determining this question, and if you find from the evidence that it was usual and customary in defendant's profession—in certain cases—to dress the arm in the extended form, and so, also, it was usual and customary to dress it in the flexed form, or in a flexed position, then you would be warranted in finding that the doctor had a right to exercise his judgment, under all the circumstances, in determining which form of treatment was the most desirable under those circumstances. If the doctor did exercise such ordinary care, in determining which was the

most desirable position for the arm, under the circumstances, then, gentlemen, the plaintiff would not be entitled to your verdict.

"The skill to be exercised by a physician, under such circumstances, should be proportionate to the character of the injury treated. If the injury treated by the surgeon is of a serious character, requiring close attention; requiring great care of him; requiring great skill; it is the duty of the physician to exercise such greater skill, such higher care, and devote more attention to such a case.

"Now, in this case, look to the character of the injury. What was the nature of it? Was it a serious injury, or otherwise? Determine from the evidence what skill was required under the circumstances; what diligence was required of a physician possessing ordinary skill and care in his profession.

"Now, it is claimed by the plaintiff, that the defendant, the doctor, was careless and negligent in not setting up passive motion in the boy's arm, prior to the time of his discharge. It is claimed by the plaintiff, that had such motion been set up in the arm, it would not have been injured permanently, and the boy would now have the proper use of his arm.

"In determining this question, gentlemen, you are to consider all the evidence, as to what time, under the circumstances, it was proper in that profession to set up the passive motion of the arm. Does the time differ in different cases? Is there in that profession, in the exercise of ordinary care, any particular time at which a physician ought to set up the passive motion? Look to the evidence, gentlemen, to determine this question; and, if you find from the evidence that the doctor was careless and negligent in not setting up passive motion at the proper time, then and in that case you are warranted in finding a verdict for the plaintiff; if you find, at the same time, that the failure to do so was the cause of the injury to the boy.

"Carefully consider all the evidence in the case bearing on that point—the medical testimony, and also the other testimony in the case.

"If you find in that particular regard that the doctor did not use the ordinary skill, care, and attention usually exercised by members of his profession in that vicinity, then plaintiff would be entitled to your verdict.

"It is claimed by the defendant, Dr. Kurtz, that he gave instructions to certain members of the family of the plaintiff in regard to setting up this passive motion, that such instructions were given to those who had charge and care of the boy, Howard Whitney.

"Now, gentlemen of the jury, if you should find from the evidence that such directions were given to those who had the care and charge of the boy; and you shall find, at the same time, that such instructions were reasonable; and shall find, at the same time, that they were not complied with by those having the boy in charge; and you shall find, at the same time, that their failure to obey such instructions materially contributed to the injury the boy has received, then, in that case, plaintiff would not be entitled to recover, even though you may find the defendant, the doctor, was negligent in his treatment and care of the arm.

"Now, gentlemen, you may take this case, and if you find that the defendant, Dr. Kurtz, has been negligent and careless in the particulars claimed by the plaintiff, and if, at the same time, you find plaintiff injured thereby; that his arm was stiffened and deformed thereby; and if, at the same time, you find that those having him in charge did not, by their omission to follow their instructions from the doctor, materially contribute to the plaintiff's injury, then, gentlemen, in that case, plaintiff would be entitled to your verdict.

"But if, on the other hand, you find the doctor not negligent and careless, as claimed here by the plaintiff, your verdict should be in favor of the defendant; and if you find that the defendant, the doctor, made a mistake of judgment, but that in making such mistake of judgment he was acting with the ordinary skill and care of his profession, then, in that case, plaintiff will not be entitled to your verdict.

"Gentlemen, if you find the defendant was negligent, but you further find that the omission and negligence of those

having care of this boy materially contributed to the boy's injury, the plaintiff would not be entitled to recover in this action.

"Should you find a verdict for the plaintiff, it will be your duty to assess his damages, and in assessing plaintiff's damages take into consideration the loss of power to use his arm and hand, if any such loss of power has been here proven; you will take this into consideration, as well as for any suffering endured by plaintiff in consequence of the want of care and skill on the part of the defendant. Plaintiff is not entitled to recover for the suffering directly occasioned by having the arm broken, and the bones of it fractured, but is only entitled to recover damages for such suffering and pain as he has endured, in addition to the other pain, consequent upon the negligent and careless treatment of him, plaintiff, by the defendant.

"Now take this case, gentlemen, and give it a full and fair consideration, and if you find for the plaintiff, assess his damages; if you find in favor of the defendant, your verdict will simply be for the defendant."

After being out less than one hour, the jury returned with a verdict in favor of defendant.

Thus ended the first civil suit with action for damages, for alleged improper treatment of fracture of the lower end of humerus by the extension method. Before the trial I confess to having felt great uneasiness. All the principal authors were emphatically against me. Stimson, one of the latest authors, had only faintly approved it, though he had never given it trial. Gross had said in his last edition: "I see no reason to change my mind upon the subject." Erichsen and Hamilton had not heard of it, and Packard mentioned it only to advise his own plan in preference. Agnew alone approved it.

The victory is a far-reaching one. It puts the stamp of approval upon a plan of treatment that has won its way against great opposition in our schools, and which, until now, has been employed at the risk of the surgeon.

HYSTERICAL RAPID RESPIRATION, WITH CASES; PECULIAR FORM OF RUPIAL SKIN DISEASE IN AN HYSTERICAL WOMAN.

By S. WEIR MITCHELL, M.D., LL.D.

[Read December 7, 1892.]

It is some years since first I called attention to the subject of the rapid breathing of certain hysterical patients. Since then I have seen it often—indeed, within six months four times. Several of these cases were of unusual value because long under my care, or so situated as to admit of graphic representation of the respiration types.

The phenomenon must be rare, as I find no allusion to it since my own paper, in 1883. A male case, by E. Bischoff,[1] reported in 1874, has to me the look of hysteria, but such can hardly be said of Brinton's and my own male case,[2] in which the rapid breathing was the reflex result of traumatism of the lung. This extraordinary case is still alive. In both of the cases just referred to, the type of respiration, though rapid, was deep and apparently laborious at times, so as always to lack the effortless regularity and seeming naturalness of the rapid hysterical breathing.

I have now seen so many of these cases connected with hysteria that I am able to formulate as to them certain conclusions.

The breathing is largely upper costal, sometimes exclusively so. It is exceptional to find the relative share as between chest and diaphragm preserved. As a rule, the breathing is slight in

[1] Deutsche Arch. f. klin. Med., vol. xii. p. 262.

[2] Trans. Phila. Coll. Phys., 1870.

amount—that is, superficial, but without appearance of effort. Usually, or early in the case, the patient is ignorant of the existence of the symptom. When this knowledge is once acquired the respiration rate is increased by excitement, even by the mere approach of nurse or doctor. In certain persons the symptom occurs only just after sleep, and may be quite absent in sleep—indeed, it is usually so. In some cases this symptom is almost the only distinct expression of hysteria, or is not present at all until the patient is emotionally excited. The number of respirations goes up and down rapidly, and without any marked coincidental change of pulse. This type of breathing is not a possible voluntary product. The effort of a healthy person to breathe as fast as these patients breathe causes exhaustion, and the graphic record is irregular and unlike that of hysteria. (See tracings.) Cases in males are more rare, and cannot always be with certainty regarded as hysterical. I shall speak of them further when quoting the cases referred to by Bischoff and that of Brinton and the author.

Dr. Coates[1] has reported a number of cases of rapid breathing. They were all people who believed, or were made to think, they had diseased lungs. From the attention they were thus led to give to these organs arose types of rapid breathing.

Case I., a woman, aged thirty years, had rapid respiration from presumed pulmonary malady. By making her count twenty without inspiring she was led to take a deep breath.

Case II. is unimportant.

In Case III., a girl, aged nineteen, there was presumption of tuberculosis, but no real lung disease. Her breathing was shallow and rapid.

Case IV., a girl, aged sixteen, was enabled by deep and abrupt quick breaths, to simulate for a time the strong, lifting impulse of cardiac hypertrophy. Two other cases are given, but in none is the number of respirations mentioned.

In 1883 Dr. E. Mackey[2] described a girl, seventeen years old, who, while sitting up in bed, breathed from eighty-eight to

[1] W. M. Coates: Brit. Med. Journ., July 5, 1884.

[2] Edw. Mackey: Lancet, Feb. 10, 1883.

ninety-three times per minute, with now and then convulsive gasps. Her pulse was feeble, and beat sixty-four times to the minute. Temperature, 99° F. She had had "chlorotic anæmia" and a loud systolic basal bruit, which was still so loud as to mask all other chest sounds. Morphine and atropine overcame the peculiar respiration symptom, but on the third day it rose to 88–128, and thereafter the case displayed a large variety of hysterical symptoms. The respiration was shallow and short.

The author calls attention to the difference between this and the rapid but deep breathing of certain cases of diabetic coma.

Dr. Bristowe,[1] amongst other hysterical disorders, seems to speak—if I do not misapprehend him—of hysterical dyspnœa with asthmatic symptoms. In one case there was rapid breathing, cough, and bloody expectoration, with no rise of temperature. He goes on to say that simple excess in the rate of breathing may be seen in hysteria. In the case of a woman, thirty or forty years old, with other hysterical signs, there were spells of rapid breathing, seventy or eighty respirations to the minute, and lasting from a few minutes up to several hours—the pulse remaining normal in frequency.

In my *Lectures on Nervous Diseases*[2] I gave several illustrations of this curious phenomenon. It is only necessary to refer to them. The one male case of rapid breathing there stated was seen by Professor Brinton and myself, and is probably alone in the records of medicine as a case of rapid breathing (50–125)—caused by bullet wound of chest. The quick respiration is said to have come immediately upon the wounding of the lung. The constant dyspnœa, the type of breathing, which was normal, and other features set this case apart from all others I have seen. Certainly it was not hysterical. In the same lectures I called attention to the diagnostic value of *rapid upper costal* respiration as sometimes of use in arousing suspicions as to the presence of hysteria in conditions of disease which would not otherwise suggest its presence. In some

[1] Bristowe: Lancet, June 20, 1885.

[2] Second ed., p. 198 et seq.

forms of insanity we may have rapid breathing, but the type has, in all such cases seen as yet by me, been normal and not merely costal, or with scarcely visible abdominal movement. Thus, in wild mental excitement from any cause, and in acute mania, the respiration may rise to forty or higher; but, as a rule, the heart is then also in too rapid motion and the chest movements are merely exaggerations of the normal action, and never, I believe, peculiar like those seen in hysteria. I saw but recently another illustration of the diagnostic fact just stated. A lady, aged sixty-two years, consulted Dr. John K. Mitchell, complaining of the following symptoms: Passive refusal to speak; no aphonia; great feebleness; absolute anorexia; much flushing of the face. I was inclined to regard the case as one of the forms of melancholia, until Dr. Mitchell called to my attention the fact that always on awaking, or from the excitement of a visit, her respiration rose at once to 50 or 60 without rise of pulse. The breathing was upper costal. He was inclined to think that hysteria was the dominating element in her case, and although I did not at first agree with him, his conclusion proved finally to be the correct one.

Some of the most puzzling diagnoses we are called upon to make are those in which a serious fall has left the patient palsied or unconscious. Occasionally the rest of the symptoms, which appear so grave, are betrayed as hysterical by the rapid rate of the respiration, a theory so constantly overlooked, that, save in acute pulmonary diseases, it is rarely stated. I have seen very lately, in consultation, a lady who suffered from the fall of a block of wood on her head. She dropped insensible, and remained thus for several days, her condition causing great alarm to her friends and immediate medical attendant. The pupils were largely dilated. There was no apparent palsy. The insensibility was not profound, but to appearance deepened slowly for several days. She awoke from this state at the end of a week, and thereafter had frequent vomiting; fixation of the head—it could be turned from side to side, but not bent backward. The upper cervical region was sensitive, and pressure caused vomiting. The pulse

was 80; the muscle reflexes all in excess. Sensation was normal in all its forms. Meanwhile there was constant headache, but healthy eye-grounds. Many things in this collection of symptoms puzzled me. The case had very little of that look of hysteria on which one gets used to relying; but the respiration was 50, and upper costal, and my decision, that all the phenomena were hysterical, has since been amply justified by the developmental changes of the case.

The following very remarkable case I give in full from Dr. Burr's notes:

Case I. *Case of hysterical knee-joint; relief; relapse; long trance condition; alleged fast of sixty-four days; recovery; aphonia; rapid respiration; unusual form of skin disease; failure of hypnotic treatment.*—The patient, a female, single, now twenty-four years of age, was first brought to the Infirmary for Nervous Diseases in November, 1883. The following notes were then taken:

Family and personal history negative. When fifteen years old she fell on her left knee while playing in the yard. She was carried into the house, put to bed, and for six weeks suffered much pain. The pain gradually disappeared, but she was unable to walk on the affected limb, and wore a bandage to support the knee. By this means she was able to walk with comparative ease. After a few weeks she removed the bandage. Almost immediately she fell, injuring the knee again. After the fall she was unconscious a short time. She was kept at perfect rest in bed for a month, the knee bandaged, and splints applied. For two years she suffered much pain, and was unable to do any work. She walked with crutches. Soon after this she had a violent attack of "hysteria." Dr. Halberstadt, of Pottsville, was called to see her. Dr. Halberstadt writes me: "When I was called to see Miss C. she complained of intermittent pain in the head, left eye, and foot, ears, and left thumb. The eyes were sensitive to light. Arms and legs rigid. Marked sweating. Respiration was 180, and pulse so rapid that I could not count it. This I saw continue for two hours, but her mother declared it had been going on for five weeks. The whole body moved at each inspiration, and her appearance was that of being worked by machinery. I could detect no disease of the kidneys or uterus." At this time she was seen by a physician from Philadelphia, who pronounced her case hopeless. She slowly improved, and finally could walk with the knee bandaged.

Her condition on admission to the Infirmary, in November, 1883, was as follows: No wasting, except that the lower third of right thigh measured five-eighths of an inch more than the left, and the right knee one and three-eighths inches more than the left. The temperature was the same on both sides. The electrical reactions were normal. Dr. Mitchell pronounced the case to be one

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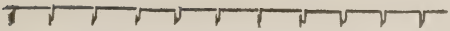
Drawing of unusual form of disease of skin
in a case of Hysteria.

TRACINGS (CASE I.).

Attempt to simulate rapid shallow breathing by a man in health.

The lower lines mark seconds, the upper the respiration curves. Rate, 60 per minute.

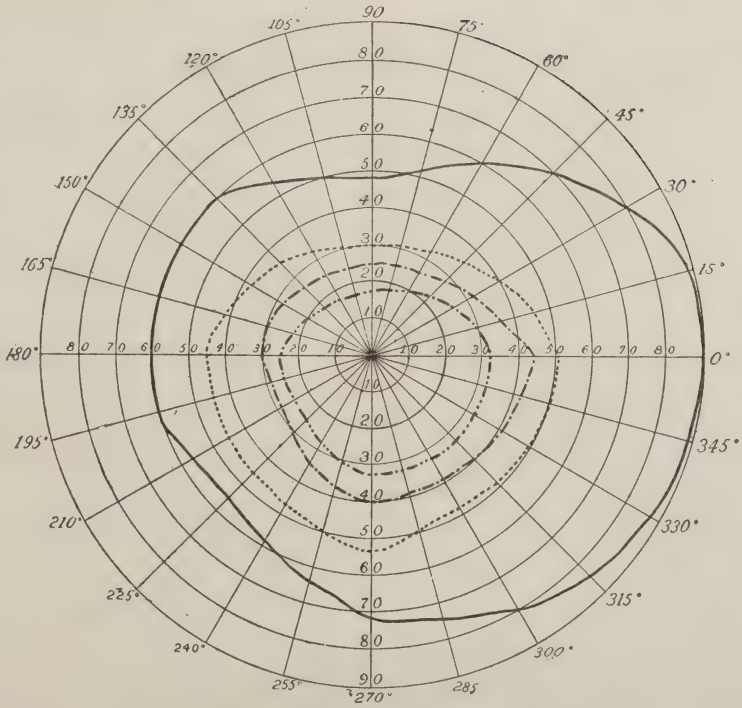
1.

2. 

1. Respiration (126 to the minute).
2. Time (seconds).

Tracing showing hysteric cough.

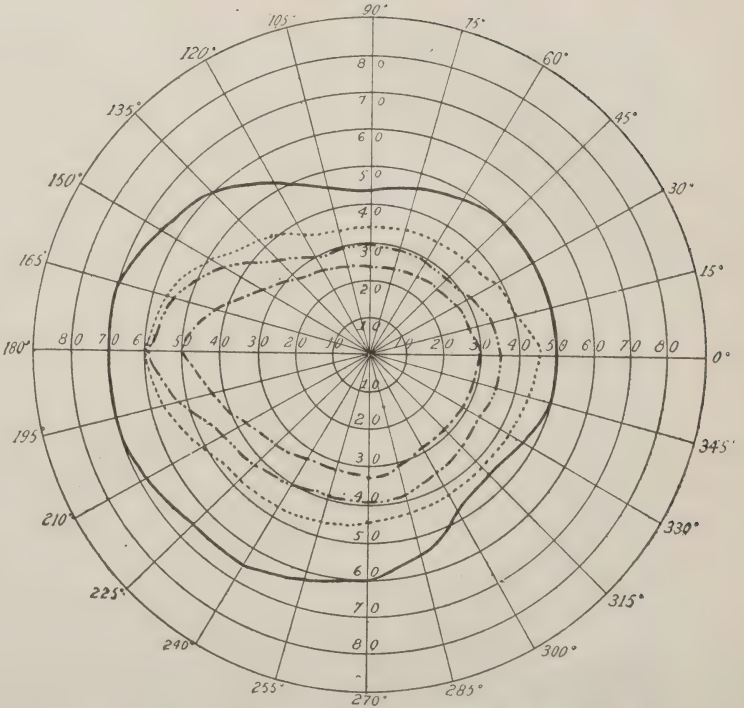
DIAGRAM I.



Field of vision of right eye for white, blue, red, and green. The outer continuous line indicates the limit of the form field; the broken lines the limits of the color fields, which are concentrically contracted.

White, ———— Red, — — — — —
Blue, ... Green, — · — · — · —

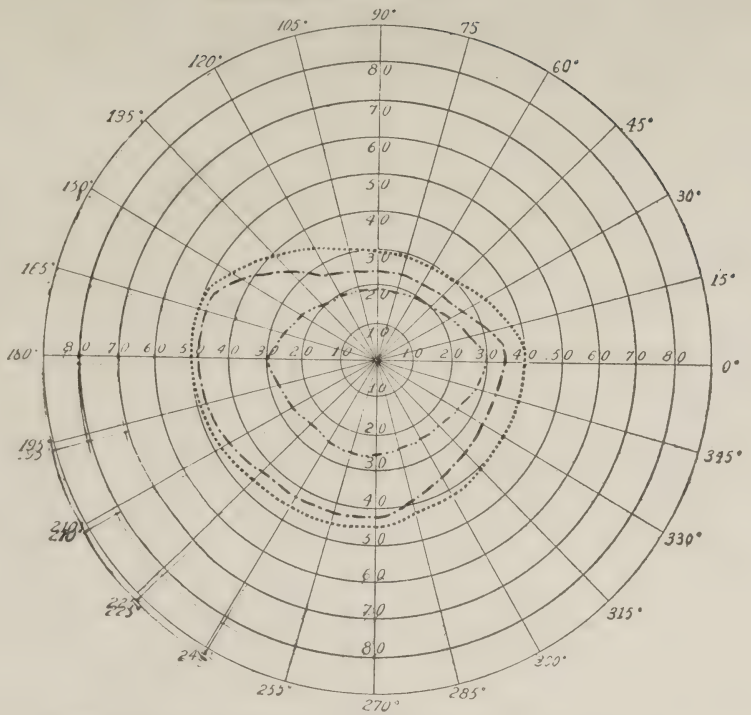
DIAGRAM II.



Field of vision of the left eye for white, blue, red, and green. The outer continuous line indicates the limit of the form field; the broken lines the limits of the color fields. There is contraction of both form and color fields, the form field having suffered proportionately the greater contraction.

White, ———— Red, — — — — —
Blue, ... Green, — · — · — · —

DIAGRAM III.

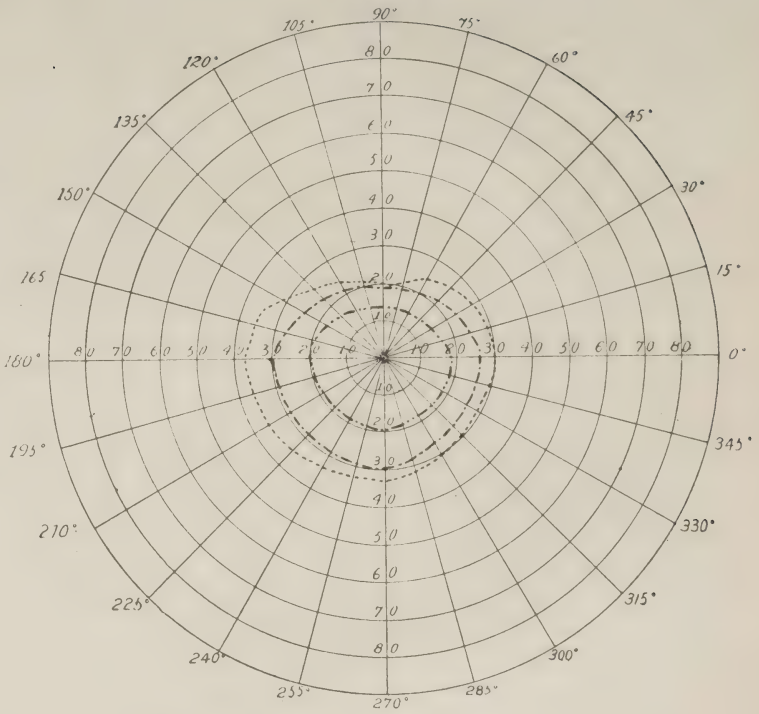


Field of vision of the left eye for blue, red, and green. The broken lines indicate that the limits of the color fields are contracted. The blue and red lines coincided in the horizontal meridian of the temporal side, but this is not accurately shown in the diagram.

Blue, ... Red, — — — — — —

Green, — — — — — —

DIAGRAM IV.



Blue, ... Red, — — — — — —

Green, — — — — — —

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of hysterical knee-joint. After one month's treatment with massage and faradism she was discharged cured.

Examination made on readmission to the Infirmary, March 22, 1892. Patient says that on leaving the Infirmary she still limped. In the autumn of 1888, on the left leg, in the place where the ulcer now is, small pimples appeared after the application of a "strengthening wash." On being told, July 7, 1889, that her father was dead, she fell into a "trance," which lasted into October. It is alleged by her family that during this time she was watched night and day. Her sister states positively that she received nothing to eat or drink during the trance, except that after the beginning of September she swallowed small quantities of water. She was given an injection weekly, which was always followed by a natural stool. She passed no urine. The bed was never even moist. The eyes were shut, and resistance was made on raising the lids. Respiration could be detected only on the closest inspection. She never moved nor even winked. Her face was yellow, but she did not lose flesh. Three or four times daily she would throw up large quantities (sometimes enough to stain eighteen towels) of a dark-reddish fluid containing clots. The faradic battery was used on the arms and legs for five weeks without effect. Until five weeks before awaking, people had been permitted to see her, and she was, indeed, on exhibition. After this was stopped she one day suddenly, without known cause, awoke, crying bitterly. She denied all recollection of what had occurred during the "trance." The left arm and leg were powerless. She could not feel the faradic battery when applied to that side. Speech was whispering. While in "trance" the pimples spoken of above, ulcerated, became confluent, and a thick crust formed.

Present state. Well nourished. Respiration varies from 120 to 150 per minute. It is shallow, almost entirely upper costal, and perfectly regular in rhythm. If the nose and mouth be held closed the respiratory movements continue, and after about one minute she makes one deep inspiratory effort. During sleep respiration falls to 18 or 20. The rate is increased when she knows she is under observation. She has frequent barking cough. The pulse averages 100 per minute. There is no wasting. When in bed she can move the left leg perfectly well against resistance, but drags the left foot when walking. Station is good. Knee-jerk is plus. Clonus is absent. The plantar reflex is marked. The elbow-jerk is present. Dynamometer: R., 95; L., 70. She is right-handed.

The tactile sense is hard to determine on account of the slight reliance that can be placed on her statements. The left side is more sensitive to pain than the right. Covering a large part of the anterior aspect of the leg from above the ankle to below the knee is a thick, broad crust much resembling the bark of an old tree. It is dark-gray and much fissured. (See colored plate.) On removing the crust, while the patient was hypnotized, there was found under it a grayish-white fibrinous material, from the surface of which oozed a little blood. Around the edges the skin was thickened, somewhat

hardened, and hyperæmic. The area was very sensitive. (The crust is in the museum of the College.)

Dr. de Schweinitz examined the patient's eyes and reported: "Conjunctiva insensitive; pupils normal in reaction; good fusion power; no lesions in the fundus oculi. In the *right eye* the form field is normal in extent; there is concentric contraction of the color fields, but they occupy their normal position. In the *left eye* there is considerable contraction of the form field, and in the horizontal meridian of the temporal side the red and blue lines of the color fields coincide. This may be seen in Diagrams I. and II., and also in Diagram III., representing the color fields alone, and taken one month later. In this chart it is evident that there is slight increase in the contraction of the color fields, but in other respects it is closely similar to the other diagrams. The field of vision for colors taken during a semi-hypnotic condition, shows that there was no material difference in color-sense of the patient during this and the normal state, except that the color field is markedly contracted and that the blue and red lines practically coincide in the vertical meridians both above and below. This may be seen in Diagram IV."

Physical examination reveals nothing abnormal in the thoracic or abdominal organs.

Remarks.—Both Dr. Burr and I repeatedly hypnotized this woman, but neither he nor I was able to see any good result. She became, under hypnotic influence, insensible to pain, and I was then able to remove from her leg the accumulated crusts. I hoped to get her, while hypnotized, to tell me the mode in which she had carried on her deceit as to her fasting, but I failed entirely. The tracings obtained for me in this case show very well the speed of breathing. (See tracings). The partial influence of an order to breathe deeply, which, in the waking state, had no effect, was well seen in one of my tracings, which has been, unfortunately, mislaid. I have added the curves obtained by the efforts of one in health to breathe like the patient. Their irregularity as contrasted with the forms of the hysterical curves of breathing is very interesting. I did this case no good whatsoever, because of her being in a ward where she was the subject of not unnatural curiosity.

Prof. Duhring's examination of the skin disease completes this interesting record; it is placed at the close of this paper. A careful search leads me to agree with Dr. Duhring in regarding this form of skin disease as of most unusual type.

Case II.—S. M., female, aged twenty-one, single, mulatto. Applied for treatment at the Infirmary September 10, 1891. There is no obtainable family history, and, owing to the woman's want of intelligence, her own symptoms and past history are inadequately related.

In 1887 she is said to have had a sharp bronchitis which lasted all winter and was accompanied with loss of voice for a year. This was probably hysterical, since in December, 1887, she suffered with colic, out of which arose a seven months' siege of varied hysterical symptoms, with frequent severe headaches and numerous convulsions of grave hystero-epileptic type. She was seven months in bed, but knows little of what passed. In May, 1888, she was taken to St. Luke's Hospital, New York, in a comatose state, and so remained four days. Having improved somewhat, she went home, where soon again she became wildly hysterical. Fits of severe character and two hours' duration were followed by stupor lasting many hours. This condition was present up to the time of her admission to the Infirmary.

At this date Prof. Hirst reports her generative organs normal, except that the womb is rather undeveloped. There is no ovarian tenderness and no evidence of epilepto-genetic spaces there or elsewhere. She has imperfect pain-sense on the left side—leg, arm, and body—with paresis of both legs and the left arm. The abdominal and thoracic viscera show no signs of disease. Appetite and digestion are normal. There is no anæmia. The knee-jerk is markedly increased. There is slight clonus—from three to five jerks. The pulse is normal; temperature normal. Respiration varies from thirty to fifty per minute. It is of upper costal type chiefly, but at times the abdomen and lower ribs move. Generally it is difficult or impossible to distinguish the least motion in the diaphragm. During sleep the respiration rate falls to twenty per minute. These peculiarities of breathing were not known to the patient. They seemed to cause no fatigue, although to breathe voluntarily as she did, very soon produces exhaustion in the healthy. She improved rapidly as to all her hysterical symptoms except the breathing.

Case III.—C. M., female, aged nineteen, single. At the age of seventeen years the patient had, after a slight accident in driving, some tenderness in the spine, and after a year, upon a fright after exposure in a thunderstorm, partially lost her voice. This was at times better, or well, but in March, 1890, she had larnygitis, with a sharp attack of the grippe, and then abruptly lost all voice except power to whisper. She was told in New York that it was not hysterical, but a rare form of loss of power in the abductor muscles of the larynx. Electricity gave no relief. The aphonia improved in the late summer. It is said to be nearly well, but the voice is easily tired. Up to November 23, 1891, when I first saw her, she had been very emotional and hysterical; tears and attacks of rigidity continued to trouble her. At this time she is in good flesh, rosy, and to appearance well, but relies much on her mother, and is at all times easily made hysterical. Her blood is close to normal in number of corpuscles and amount of hæmoglobin. All the diges-

tive, renal, and menstrual functions are well performed. There is no ovarian tenderness.

Sensation. She is over-sensitive to a pinch of the muscles. The arms, back, and abdomen, but not the face, present small, analgesic areas, irregular in form, one to two inches wide, and varying in location from day to day. There is no loss of sense of touch, locality, or temperature. The legs present no analgesic areas. There is pain in the back along the spine and over the loins. It is worse on exertion, and at times absent. There is constant severe pain in the posterior aspects of both legs in the gluteal regions. This is apparently a muscular trouble, since in all these parts pressure is painful.

Locomotion. When supine she can move all limbs well, and both extended legs can be lifted together. When erect she sways with jerky recovery of position. With the eyes shut this is enormously increased, presenting a true type of the hysterical ataxia I was the first to describe. Thus, when erect with the feet together, for a moment all is well, then she sways forward or backward or right or left eight or ten inches, and with an abrupt effort recovers her upright position, only to fall to and fro anew. Her walk is better but is not quite regular, and she drags the left foot with the toes outward. The knee-jerk is much increased. There is slight ankle clonus—five or six movements—on the right. On the left it is absent. The bladder action is normal. All motion wearies her quickly. Her pulse is usually higher at night, but is very changeable. The respiration also varies, but on the least excitement rises and remains high for an hour or more. Massage, electricity, my visits, etc., all increase the respiration rate. Her pulse and respiration averaged as follows:

Respiration, 40	Pulse, 80
" 30	" 75
" 60	" 85

She was put at rest, forbidden to speak for three weeks, and given localized faradic currents and daily massage. She made a good recovery, but only after some months won complete use of all her powers.

I have seen, as I have said, other cases and many of this type of rapid hysterical breathing, but the cases here given may suffice.

Dr. Duhring: I had the good fortune, through Dr. Mitchell, to see Case I. a few weeks ago. The disease had existed three or four years, and there were several remarkable points about the crust. It differed in character from the known crusts, as those of late syphilis, and those from simple ulcer. It differed from syphilis in that it was made up

largely of epithelium and not of dried pus. This was verified by the microscope. This, too, was manifested on seeing the lesion of the skin proper without the crust. There was no ulceration beneath the crust, but the epithelial layer and rete mucosum were atrophied and diseased. The papillary layer was reddened and infiltrated as seen in chronic tinea favosa. The cause and origin of this lesion and the crusts must be considered together, as they are a part of the disease. The interpretation of the cause as suggested by Dr. Mitchell, namely, that it is of nervous origin, is, I believe, correct. It is due to a degeneration of the nervous system, giving rise to local trophic disturbance of the skin. We may designate it as a peculiar trophic crusted disease of the skin. The state of the whole nervous system is accountable for this disease. It is a local manifestation of a general neurotic affection, and not a disease produced by changes in a single nerve or set of nerves.

The nature of the disease, pathologically, is an inflammation of the skin and subdermal tissue of a low type, due to aberrations of local nutrition. Sarcoma and other similar ulcerative or crusted diseases are not suggested by the appearance of the lesion. It is very difficult to class such cases in dermatology. It bears some resemblance, from a pathogenic standpoint, to keratoma and callosity. The disease may be grouped with such cases as that of the one described and portrayed in the second number of the *International Atlas of Rare Skin Diseases*, by E. Besnier, with the title "Keratodermia Erythematosa." In regard to the crusts on bromide ulcers, if they are examined they will be found to be made up of sebaceous matter and pus—they are, in fact, largely puriform in character. I saw a noted case in London, shown to me by my friend Dr. Tilbury Fox. I have a photograph of it. The crusts were the size of the hand and were made up of sebaceous matter and pus.

APPENDIX.

ADDITIONS TO THE MÜTTER MUSEUM, 1892.

Specimens.

Colloid Cancer of the Stomach. Presented by Dr. J. K. Mitchell.

Porencephalon. Presented by Dr. James Hendrie Lloyd.

Retained Placenta. Presented by Dr. W. F. Atlee.

Remains of a Ring Pessary removed from the Vagina after Eight Years. Presented by Dr. C. W. Dulles.

Micro-photographs of the Nervous System. Presented.

Antique Stethoscope, Specula, Syringes, Pocket Case of Instruments, and Cupping Apparatus. Presented.

Vesical Calculus. Weight eight ounces. Presented by Dr. J. Henry C. Simes.

Myoma of Uterus. Presented by Dr. Joseph Price.

Cast of Chinese Woman's Foot. Presented by Dr. Thomas S. K. Morton.

Cystic Kidney. Presented by Dr. John H. Packard.

Lungs showing Anthracosis. Presented by Dr. D. J. Milton Miller.

Photograph of a tablet erected in memory of the Acting Assistant Surgeons of the United States Army who have fallen in the line of duty in the service of their country. Presented by Dr. Richard J. Dunglison.

Photograph of the blended Tocci Twins. Purchased.

Lungs showing Acute Tuberculous Broncho-pneumonia. Presented by Dr. S. Solis-Cohen.

There has been received on deposit a valuable Anatomical Collection belonging to Dr. George McClellan.

Remarkable Scab from the Leg of a Hysterical Woman; with photograph. Presented by Dr. S. Weir Mitchell.

Series of Photographs illustrating Chinese Surgery. Presented by Dr. R. P. Harris.

Forty Micro-photographs of Cases of Quinine Blindness and Neuroma of the Eyelid. Presented by Drs. George E. de Schweinitz and William M. Gray.

Uterine Fibroids. Presented by Dr. J. Ewing Mears.

Three Uterine Fibroids. Presented by Dr. W. J. Taylor.

Sixty Indian Skulls from Mounds near St. Louis, Mo. Presented by Dr. S. Weir Mitchell.

Indian Bones from Virginia. Presented by Dr. John B. Roberts.

Plaster cast of Hands showing Leprosy. Presented by Dr. A. C. A. Beecher.

Six Antique Stethoscopes. Presented by Dr. John K. Mitchell.

Fracture of the Humerus. Presented by Dr. W. G. Porter.

Head of Femur; Coxitis. Presented by Dr. W. G. Porter.

Fibroid Tumor of Thyroid Gland. Presented by Dr. W. G. Porter.

Tænia. Presented by Dr. W. G. Porter.

Illustrated Books.

Sandman, G. Tafel des menschlichen Gehörorganes. Berlin, 1892. Purchased.

McClellan, George. Regional Anatomy. 2 vols., 4to. Philadelphia, 1892. Purchased.

Jeançon, J. A. Pathological Anatomy. Twenty-five parts; 100 plates. Cincinnati, 1884. Presented by Dr. John H. Musser.

Sappey, Ph. C. Description et Iconographie des Vaisseaux Lymphatiques considérés chez l'Homme et les Vertébrés. Paris, 1885. Folio. Purchased.

Hirst and Piersol. Human Monstrosities. Philadelphia, 1892. Folio. Purchased.

MÜTTER MUSEUM OF THE COLLEGE OF PHYSICIANS.

N. E. CORNER THIRTEENTH AND LOCUST STREETS.

Philadelphia, January 1, 1893.

In order to increase the usefulness of the Museum, and to add to its already remarkable collections, it is requested that the Fellows of the College and friends of the Museum in the profession generally, and the public at large, will make it the repository of specimens which they possess, that have anatomical or physiological interest.

All preparations or specimens of Normal or Pathological Anatomy, Models, Illustrations, Microscopic Specimens, Surgical Apparatus, and Materia Medica, and all Antiquated or Modern Instruments will be gladly received. Every article donated will be plainly marked with the name of the donor.

Guy Hinsdale, M.D.,
Curator.

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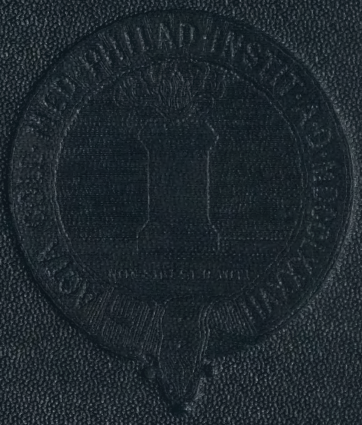
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